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SYMPOSIUM ON DIABETES MELLITUS

STATISTICS FROM OUR LADY OF THE LAKE SANITARIUM, BATON ROUGE, LOUISIANA

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INTRODUCTION

This symposium is a duplication of our prior efforts at the regular Medical Staff Meeting at Our Lady of the Lake Sanitarium in Baton Rouge, Louisiana.

We are not following the usual procedural pattern of reporting cases and then adding our summaries to the already vast total present in contemporary writings. On the contrary, this symposium originated because of the cases found at Our Lady of the Lake Sanitarium over a twenty-three month period from January 1950, through November 1951. (see Table 1).

TABLE 1
PERIOD COVERED: JANUARY 1950, THROUGH NOVEMBER 1951

Charts surveyed	20,000
Cases found	178
Primary condition	81
Associated condition	97
Average age	54 years
Youngest 7 years	
Eldest 88 years	

A total of 20,000 hospital admission charts were reviewed. Approximately 1 per cent, (178 cases), either had diabetes mellitus listed as the primary (81 cases) or associated (97 cases) cause for admission during this period. Females outnumbered males by slightly better than a 2:1 ratio. The average age was 54 years, with the youngest being 7 years of age and the eldest 88 years of age.

Table 2 lists the surgical admissions according to specialties concerned. General surgery, (primarily vascular), and ophthalmologic surgery, (primarily lens) outnumber the gynecologic and obstetrical cases as one would expect from the advanced average age previously noted.

TABLE 2
SURGICAL ADMISSIONS

General surgery	19
Ophthalmology	10
Gynecology	4
Obstetrics	3

Table 3 shows that acidosis or coma or both are not too common. It is of interest to note the number of patients having associated cardiac conditions. It is more interesting to note that despite the original 2:1 female to male ratio noted, cardiac complications affected 40 per cent of the males and 39 per cent of the females.

TABLE 3
MEDICAL COMPLICATIONS

Acidosis	10
Coma	7
Acidosis and coma	2
Associated cardiac condition (40% males, 39% females)	70

Table 4 gives the total mortality for the 178 cases found for this survey. Complications produced by the disease are apparently three times more deadly than diabetes mellitus itself.

TABLE 4
MORTALITY

Total deaths	17
Diabetes mellitus—primary cause	4
Diabetes mellitus—secondary cause	13

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PATHOGENESIS OF DIABETES*

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The exact mechanisms of human diabetes are at present unknown and any discussion of them is by necessity speculative.

In experimental diabetes the manifestations are a reflection of the state of the beta cells of the islands of Langerhans. This type of diabetes may be produced in five ways: (1) partial pancreatectomy;[1] (2) injection of anterohypophyseal extract (A.P.E.);[2] (3) partial pancreatectomy followed by administration of thyroid extract;[3] (4) administration of alloxan;[4] and (5) injection of large amounts of glucose.[5] In these cases the beta cells undergo degranulation followed by hydroptic change, cytolysis and disintegration of the cell.

In human diabetes, by the use of special stains, Bell[6] has demonstrated complete degranulation of beta cells in 25 per cent of cases and a partial degranulation in an additional 35 per cent. Equally important is that 40 per cent of the cases show no alterations from normal. Hydroptic changes do not follow degranulation in human diabetes, and cytolysis has not been demonstrated. Diabetic cases are seen in which hyalinization of the islands has occurred (50 per cent) and Opie[7] believes this to be the lesion specific for human diabetes. This hyaline material is deposited intercellularly and is not formed in the islet cells. This hyalinization is often associated with arteriosclerosis and is found in the older age group of diabetics.

In human diabetes the average pancreatic insulin content is 0.4 units per gram. The normal average pancreas contains 1.7 units of insulin per gram. The diabetic pancreatic values vary from 0.03 to 1.9 units per gram and the normal pancreas values vary from 0.6 to 3.8 units per gram.[8] When divided into age groups and with comparison of granulation of beta cells, a direct correlation of the insulin content and state of granulation is seen (Table 1).

TABLE 1
INSULIN CONTENT COMPARED WITH DEGREE OF GRANULATION

AGE GROUP	U/GM. INSULIN	AVG. DEGREE OF GRAN. OF BETA CELLS
1 - 30 yrs.	0.07 u/gm	0 - 1
30 - 60 yrs.	0.45 u/gm	1 - 2
60 - over	0.544 u/gm	2 - 3*

* = complete granulation.

With this information, we can compare experimental and human diabetes (Table 2).

As seen from Table 2 there can be no true comparison between experimental and human diabetes. Consequently we must look further for the cause and consider the endocrine glands.

In relationship to insulin and the pancreas, three possibilities exist; (1) insulin is not being formed in adequate amounts by the beta cells (degranulated); (2) normally formed insulin is not released from the pancreas in adequate amounts (fully granulated); or (3) normally formed and normally delivered insulin is neutralized or excessively metabolized after reaching the general circulation (degranulation due to overstimulation).

When the liver of a previously depancreatized dog is removed there is just as rapid a fall in the blood sugar level as following hepatectomy in a normal dog (Fig. 1).[10] This fact tends to substantiate the overproduction theory of diabetes against the nonutilization theory. This is visually illustrated in figure 2[11] where hepatectomy would produce hypoglycemia in 4 (a) and 4 (c) but not in 4 (b).

A deficiency of anterior pituitary hormone will produce a hypoglycemia, and conversely, administration of anterior pituitary extracts will maintain a high blood sugar level.

The adrenal cortex and thyroid gland, like the pituitary from which their action is separated with difficulty, will in turn lower or raise the blood sugar level similar to the pituitary gland.

The relationship then of the pancreas, pituitary, adrenal cortex and thyroid gland

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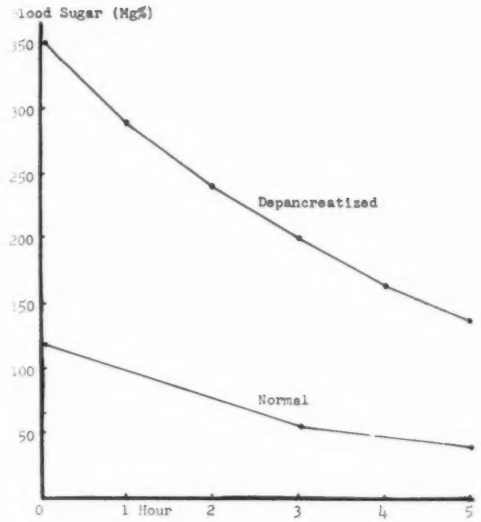

Figure 1.—Blood sugar level in normal and depancreatized dog. (From Mann, F. C., and Magath, T. B.: Arch. Int. Med. 31:797, 1923).

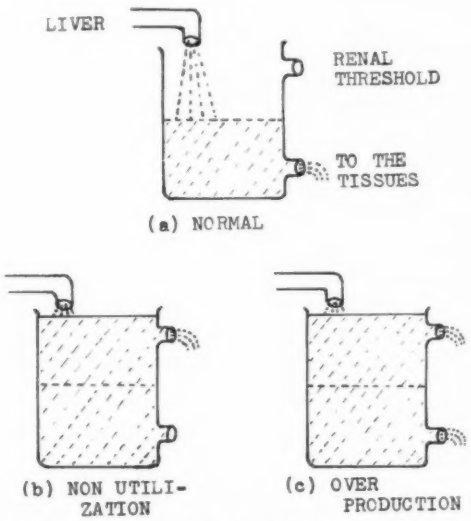

Figure 2.—Theories of diabetes (graphic) (From Soskin, S.: The endocrines in diabetes, Am. Lect. Series, No. 19).

to carbohydrate regulation and to each other is a finely balanced adjustment to supply the correct amount of glucose that is being utilized by the tissues. This is illustrated in figure 3.[11] A weakening in any direction will produce the diabetic syndrome. It may be corrected by adjustment in the opposite gland or administration of the specific deficiency, be it insulin, thyroid, adrenal, or pituitary extract.

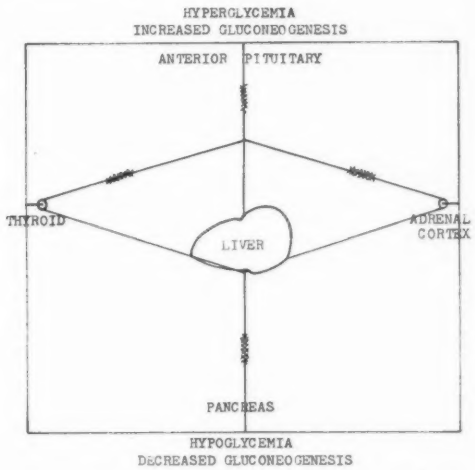

Figure 3.—Carbohydrate metabolism regulation. (From Soskin, S.: The endocrines in diabetes, Am. Lect. Series, No. 19).

SUMMARY

In summary, the condition of diabetes mellitus is a complex hormonal interplay of carbohydrate metabolism and not a simple pancreatic change producing hypoinsulinism. This understanding is essential for the modern day treatment of diabetes and it is not too much to hope that as our knowledge of these basic hormone activities and interrelationships increases the reason for the widespread incidence of diabetes in man will be revealed.

REFERENCES

1. Allen, F. M., Jr.: Metab. Rev. 1:1, 1922.

2. Young, F. G.: Lancet 2:372, 1937.

3. DeFinis, H. L. and Houssay, B. A.: Rev. Soc. Argent. Biol. 19:94, 1943.

4. Jacobs, H. R.: Proc. Soc. Exp. Biol. & Med. 37:407, 1937.

5. Doham, F. C. and Lukens, F. D. W.: Science, 105:183, 1947.

6. Bell, E. T.: Experimental Diabetes Mellitus—1st Ed., 1948. Charles C. Thomas, Springfield, Ill.

7. Opie, E. L., Chap. in Spec. Cytology, N. Y. 1928, edited by Cowdry.

8. Scott, D. A. and Fisher, A. M.: J. Clin. Invest. 17: 725, 1938.

9. Peterson, C. A.: Bul. Univ. Minnesota Hosp. & Med. Found., 21:8, 1949.

10. Mann, F. C. and Magath, T. B.: Arch. Int. Med., 31:797, 1923.

11. Soskin, S.: The endocrines in diabetes, Am. Lect. Series, No. 19.

TABLE 2
MORPHOLOGIC COMPARISON OF EXPERIMENTAL AND HUMAN DIABETES

Experiment	Beta Granulation	Beta Cells	Insulin Content	Probable Mechanism
Non-Diabetic:				
Fasting			Reduced	Resting
Fat-feeding	Reduced	Degranulation only	Reduced	Resting
Insulin inj.			Reduced	Resting
Glucose I.V.			?	Exhaustion
Temp. Diabetic:				
Hypophyseal (A.P.E.)	Reduced	Degranulation Hydropic Degen.	Reduced	Exhaustion + toxic
Thyroid				
Perm. Diabetic:				
Meta-pancreatic				Exhaustion
Meta-hypoph.	0	Necrotic Disappear	Reduced	Exhaustion + toxic
Meta-thyroid				Exhaustion + toxic
Meta-alloxan				Toxic
Meta-glucose?	Reduced	Degranulation Hydropic degen. Cytolysis	?	Exhaustion
Human Diabetes	Normal —40% Reduced—35% 0 —25%	Degranulation only No hydropic. degen. No cytolysis	¼ of normal	?

(After Peterson, C. A.: Bul. Univ. Minnesota Hosp. & Med. Found., 21:18, 1949)

DIABETES MELLITUS; OPHTHALMOLOGICAL ASPECTS

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The disease complex known as diabetes mellitus may be accompanied by pathological manifestations in almost any of the structures of the eye. For reasons of simplicity these will be treated in three groups: (1) the lens, (2) the retina, and (3) all others.

THE LENS

In spite of considerable confusion to the contrary the only type of cataract that justifies the term "diabetic cataract" is a rapidly progressive one, found in people less than forty years of age and whose onset is characterized by the presence of a layer of small punctate opacities in the anterior and posterior cortex. The great majority of the cataracts seen in diabetes are of the senile variety and are associated with diabetes only by chance. It has been repeatedly pointed out that a large percentage of apparently normal people over sixty years of age have some type of lens change. Therefore, it is difficult or impossible to evaluate the role of diabetes in the production of senile changes. However, it is the feeling of most observers that senile cataract occurs more frequently at an earlier age and progresses more rapidly to maturity in the diabetic than in the otherwise normal individual.

It will be interesting, I believe, to look at some figures on the occurrence of cataract in diabetes. In his monolithic work on cataract Kirby quotes some figures that were earlier compiled by himself. Thirty-five per cent of diabetics of all ages had clear lenses, the remaining 65 per cent hav-

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ing some variety of lens opacity. Seventy per cent of these were of the senile cortical type, 21 per cent nuclear, 7 per cent posterior cortical, and 2 per cent subcapsular. The severity of the diabetes made little difference in the incidence, cataract actually being more frequent in the moderate than in the severe cases. Approaching these figures from the standpoint of duration of the disease it is found that in diabetes of one year's duration 22 per cent had cataract, of two years' standing 62 per cent, three years' 44 per cent, four years' 63 per cent, five years' 40 per cent and over five years' 70 per cent. Also it was found that 16 per cent of diabetics less than thirty-three years of age had lens changes.

Before leaving the subject of cataract I would like to speak a word for the intense cooperation that must be obtained between the clinician and the ophthalmologist when a diabetic goes to cataract surgery. The two main things to be borne in mind are that too intense insulin therapy leads to hemorrhage, while insufficient insulin predisposes to infection. I would also like to mention the bizarre and evanescent refractive changes that occasionally occur in diabetes. All of us who do refractions have encountered the sudden loss of hypermetropia or increase of myopia that obtains at times in our diabetic patients. Various explanations have been offered. Among these are subclinical lenticular changes, changes in refraction in other ocular media, and accommodative paresis.

THE RETINA

The existence of specific changes in the fundi of diabetics was first described by Jaeger in 1856, and ever since then there has been considerable argument as to whether these changes are really specific to the disease or not. These characteristic changes are usually bilateral and are found in the area between the upper and lower temporal vessels of the retina and in the region surrounding the optic nerve head. Here numerous hemorrhages of all sizes are found. The one most often seen is small, round, and deeply situated. Also, there are exudative spots that are sharply demarcated, white or yellowish, lustrous, and give the appearance of hardness. The discs are normal and there is an absence of retinal edema. In longer standing cases there is retinitis proliferans, vitreous hemorrhage with massive organization and subsequent retinal detachment, and also, secondary glaucoma. In younger people particularly, all the changes seem to be out of proportion with the changes seen in the blood vessels.

The incidence of diabetic retinopathy has been variously reported. Elwyn quotes the statistics of Waite and Beetham who examined slightly under four thousand diabetics with visible fundi. They found deep retinal hemorrhages in 18.6 per cent, waxy exudates 10.7 per cent, nerve fiber layer hemorrhages 5 per cent, cotton-wool exudates 4.3 per cent, iridescent crystals 0.7 per cent, and proliferation of capillaries in retina 0.7 per cent. I feel that the high incidence of cotton-wool exudates, 4.3 per cent, is ample evidence to give credence to the view that arteriosclerosis as such has a large part to play in the production of so-called diabetic retinopathy. I might also note that Joslin found vascular damage in 70 per cent of 250 patients whose diabetes began in youth.

Which diabetics develop retinopathy? The older authors, and the Europeans, still emphasize that it affects the older patients only, particularly those in the 50 to 55 age group. Most American investigators do not correlate the incidence of retinopathy with either age or severity of the disease but state rather that it is seen more often in the person with mild diabetes who has had hyperglycemia for a long time. The explanation for this is the supposed chronic state of stasis due to the dilatation of the precapillary arterioles, capillaries, and postcapillary venules of the retina.

What can be done for the retinopathy? Treatment of the diabetes as such has very little effect on the retinal disease. Rutin has been advocated as a measure to diminish capillary fragility, and consequently, reduce the number of retinal hemorrhages. While used frequently the results have been

disappointing. An interesting observation was made by a group at the Cleveland Clinic. They found that patients with retinopathy have normal or slightly reduced plasma proteins with a relative decrease in albumin and increase of beta globulin. Sixteen patients were observed over a period of two to three and one-half years and given 100 to 200 grams of protein per day in an attempt to decrease the tendency to exudation and hemorrhage. Of these the plasma protein pattern was corrected and recurrent hemorrhages disappeared in 6 and decreased in 1.

OTHER MANIFESTATIONS

As stated earlier diabetes may affect any of the ocular structures. One manifestation frequently described is recurrent hordeola or even carbuncles of the lid in young unrecognized diabetics. Also there are iris changes including iritis, atrophy, rubeosis, and changes in the pupillary border. Another condition of the fundus that should be mentioned is lipemia retinalis which is associated with generalized lipemia. Finally, extra-ocular muscle palsies have been reported and also asthenopia.

REFERENCES

Berliner, M. L. Biomicroscopy of the Eye, 2, 1949, Paul B. Hoeber, Inc., New York.

Duke-Elder, W. S.: Text Book of Ophthalmology, 3, 1941, C. V. Mosby, St. Louis.

Elwyn, H.: Diseases of the Retina, 1946, The Blakiston Co., Philadelphia.

Kirby, D. B.: Surgery of Cataract, 1950, J. B. Lippincott, Philadelphia.

Schneider, R. W., McCullagh, E. P., Ruedemann, A. D., Kennedy, R. J., and Lewis, L. A.: Cleveland Clin. Quart. 14:76, 1947.

Tassman, I. S.: Eye Manifestations of Internal Diseases, 1946, C. V. Mosby, St. Louis.

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DIABETIC COMA*

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DEFINITION

Diabetic coma is a first rank medical emergency in which there is an acute insulin deficiency with resultant inability of the tissue to oxidize carbohydrate. When carbohydrate utilization is reduced to a degree which causes an increase of great magnitude of fat metabolism, ketone bodies (acetone, diacetic acid and beta-hydroxybuturic) are produced at a rate which exceeds the ability of the body to complete their oxidation. They accumulate in the blood in large quantities, and diabetic acidosis makes itself apparent.

ETIOLOGY AND PREDISPOSING CAUSES

1. *Omission of insulin* is an important cause of diabetic coma. It has been ably stated that "the diabetic who knows the most lives the longest." Every patient with diabetes, whether mild or severe, should be carefully instructed as to the benefits of insulin in order that he may recognize the urgent need for its use should his carbohydrate tolerance suddenly become impaired by infection, surgery, trauma, or an acute gastrointestinal upset. He should be cognizant of the fact that in such cases his insulin may have to be doubled or trebled instead of omitted.

2. *Infection.*—Acute infection is prone to aggravate the severity of diabetes and may precipitate diabetic coma by reducing carbohydrate tolerance and increasing fat metabolism.

Even a "trifling cold" or an upper respiratory infection may interfere with carbohydrate tolerance. A diabetic who has been maintained sugar-free on diet alone may have to take insulin in the presence of infection to avoid a serious catastophe. Therefore, I feel that the diabetic should have thorough knowledge of his disease. In order that he may do so, I furnish him with an abundance of literature on diabetes as well as inform him that he is actually his own doctor and any errors he makes will simply be chalked up as negligence or ignorance.

In the presence of infection, the urine should be examined several times daily, not only for sugar but for acetone and diacetic acid. There seems to be no need for carbohydrate restriction when infection is present, but proteins and fats should be reduced to prevent excess ketone body production.

3. *Trauma.* — Trauma very frequently has been found to precipitate diabetic coma. This is especially true in head injuries and fractures of long bones. Injuries directly

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

to the pancreas sufficient to interfere with insulin production have been spoken of, but play a minor role in the incidence of coma.

4. *Vomiting and Diarrhea.*—Any diabetic patient with vomiting and diarrhea, whether mild or otherwise, is certainly a good candidate for diabetic coma. One invariably finds that such patients will omit insulin under these circumstances, when actually it may be needed more at that particular time, not only in the amounts usually taken but even doubled to prevent a ketosis.

5. *Hyperthyroidism and pregnancy* are two separate entities that have been known to predispose not only to diabetes but to diabetic coma. The presence of either in a diabetic patient should serve as ample warning that the patient should be carefully watched for a gradual increase in blood sugar levels to the precoma and coma state.

6. *Dietetic Imbalance.*—There is evidence that production of acetone bodies will be accelerated whenever the hepatic glycogen concentration falls to subnormal levels, and such production of acetone bodies can be inhibited by any factor which will enable the liver to retain adequate stores of glycogen. The timeworn statement "fat burns in the flame of carbohydrates" has been replaced by "the flames of carbohydrate extinguish those of fat." When the physician provides the diabetic with an adequate amount of carbohydrate, he affords him his greatest insurance against coma.

7. *Insulin Resistance.*—One must remember that the diabetic whose urine is sugar-free is almost never in danger of acidosis; but in rare cases, patients have been found to be resistant to insulin, making it rather difficult at times to control diabetes with diet alone.

SYMPTOMS AND SIGNS

The symptoms of diabetic acidosis are divided into (1) early and (2) late, depending on the severity of the acidosis present. It is not uncommon to find a patient with a blood sugar of 300 to 400 mg. per 100 cc. of blood, and yet tests for ketone bodies in both the urine and plasma are negative. Therefore, one must remember, in order to avoid errors in diagnosis of coma, that the diabetic is subject to any and all of the diseases that may affect any nondiabetic. The diabetic patient may be unconscious from a fractured skull, from acute intoxication, cerebral hemorrhage, from drugs or meningitis, or from shock. All of these may alter carbohydrate metabolism.

Therefore, may I reiterate that an unconscious patient with an elevated blood sugar in the absence of ketosis should be carefully studied to determine the cause of his coma.

The early symptoms are usually vague. Acidosis should be suspected when any unusual symptoms or signs appear in a known diabetic. The most frequent early symptoms are weakness, headache, thirst, loss of appetite, vomiting, drowsiness, and neuritic pains.

The late symptoms are much more characteristic; namely, dehydration, Kussmaul breathing, somnolence, coma, and acetone odor to the breath. The cheeks are flushed, respirations are deep and rapid, the skin and mouth are dry, and the eyeballs are soft. The late symptoms begin to manifest themselves when the CO_2 combining power falls below 40 volumes per cent, and they are pronounced if the CO_2 combining power falls below 20 volumes per cent.

PATHOLOGIC PHYSIOLOGY

Before we discuss the treatment of diabetic coma, it is always wise to understand the nature of diabetic coma.

Diabetic ketoses occur when utilization of carbohydrate is reduced to a degree which causes an increase of great magnitude in the metabolism of fats. This results in the production of ketone bodies (acetone, diacetic acid, and beta-hydroxybuturic acid). They are produced at a rate which exceeds the ability of the body to complete their oxidation, and the excess, in the early stages, is excreted in the urine. As this process progresses, a 1 plus reaction for acetone in the urine is replaced by a 2 plus, 3 plus, and finally, a 4 plus reaction. Then the production exceeds the functional capacity of the excretory mechanism of the kidneys and the lungs, and then ketone bodies accumulate in the blood. At first, this stage is detectable by the appearance of a

trace of acetone in the blood plasma; but barring correction, 2 plus, 3 plus, and finally, 4 plus reactions for plasma acetone are observed. It is not until 4 plus reactions for acetone in the urine are observed for variable periods of time that amounts of acetone increase in the plasma to a sufficient concentration to be detected by the usual tests used. Hence, a 3 plus or 4 plus reaction for acetone in the plasma shows a much more advanced degree of ketosis than do similar reactions obtained on examination of the urine.

CRITERIA FOR DIAGNOSIS

It has been ably stated by Duncan, Carey and Hudson, that an acutely ill patient having a 4 plus glycosuria and acetonuria and a 4 plus reaction for plasma acetone fulfills the chemical criteria necessary to make a diagnosis of diabetic coma. With this in mind, they have instituted treatment fully one hour before blood sugar determinations were obtained from the laboratory. When one considers that the mortality rate increases with every additional hour of coma, it is imperative that we institute prompt treatment so as not to have avoidable deaths.

A plasma acetone test is simple and can be done in ten to fifteen minutes. Withdraw 5 cc. blood, allow to stand for ten minutes, take 2 or 3 drops of plasma, place on a pinch of Denco-Reagent.* In the presence of acetone, a shade of purple will develop in thirty seconds; a trace of acetone will result in a light lavender color, and with increasing amounts of acetone the color will be darker; a dark blue indicating a 4 plus reaction. In the absence of acetone a grayish yellow color will result.

The following chemistry would be noted in a patient with diabetic coma: a 4 plus glycosuria and acetonuria; blood sugar 300 mg. per 100 cc. blood or over; CO_2 combining power 25 volumes per cent or below.

TREATMENT

The treatment of diabetic acidosis has been controversial. Story and his associates state that alkalies, plasma, and potassium preparations are not necessary in the treatment of diabetic coma if adequate insulin dosage is given early. On the other hand, Duncan, Carey, and Hudson state that alkalies, plasma, and potassium are definitely of value in the treatment of diabetic coma along with adequate insulin dosage if the mortality rate is to be reduced still more.

I believe that there is little doubt at present that large doses of insulin given early in the treatment of diabetic coma have reduced the mortality rate considerably. Each case must be individualized. There are cases in which alkalies, plasma, and potassium are not necessary, and cases in which they are necessary, and their judicious use will be life-saving in severe diabetic acidosis.

Be that as it may, the following method of treatment of diabetic coma has proved satisfactory:

I. A. Secure the following studies immediately:

1. Blood sugar, acetone bodies, hematocrit, CO_2 combining power, specific gravity (of whole blood) urea determinations, and serum potassium.

2. Urine for culture and routine chemical analysis.

B. Subsequent studies:

1. Urine should be examined at hourly intervals for sugar and acetone.

2. Blood specimens should be taken at two and three hour intervals for sugar, acetone, CO_2 and specific gravity of whole blood until the patient is conscious and retaining nourishment by mouth.

II. Insulin:

The insulin dosage may vary, depending on the severity of the coma, but a patient wtih a CO_2 combining power below 25 volumes per cent and a blood sugar above 400 mg. per 100 cc. blood and a 4 plus acetonuria should be given 40 units of regular insulin, intravenously, and 60 units of regular insulin, subcutaneously, immediately. Then insulin is given at half-hour intervals, 50 units, subcutaneously, until there is a

*Denco-Reagent contains sodium bicarbonate, ammonium sulphate and sodium nitro-prusside in anhydrous form (Gradwall).

satisfactory reduction of plasma acetone, or increase in the CO_2 combining power, or a reduction of blood sugar below 200 mg. per 100 cc. blood. If after six hours, no appreciable change is noted in either the plasma acetone, the CO_2 combining power or the blood sugar, each succeeding dose of insulin should be increased by 25 units until such change is noted. Using insulin in such tremendous dosage is not without danger, so in order to prevent a hypoglycemia it is wise to give the patient 1000 cc. of a 5 per cent glucose solution. This should be begun four hours after treatment has begun. When there is an appreciable reduction of plasma acetone and an appreciable increase of CO_2 combining power to 30 volumes per cent and a reduction blood sugar below 200 mg. per 100 cc. blood, one should recognize the fact that there will be a lessened resistance to insulin, and the physician should be on the alert and watch for a rapidly developing hypoglycemia.

When the clinical condition and the laboratory findings indicate that the patient shows satisfactory improvement, insulin may be given on an hourly basis, then on a three hour schedule, using the following guide:

4 plus glycosuria—30 units
3 plus glycosuria—20 units
2 plus glycosuria—10 units
1 plus glycosuria—omit dose
0 plus glycosuria—omit dose and give 20 gm. carbohydrate

When evidence of ketosis has subsided and the patient is taking nourishment orally, he is put on a six hour schedule using the above scale.

III. Fluids and Chlorides:

Low blood pressure, hemoconcentration, dehydration, dryness of the skin, and soft eyeballs are indicative of the shock syndrome in severe diabetic coma. Restoration of the electrolyte balance of the patient in coma is an essential factor and should not be delayed. Therefore 2000 cc. of normal saline are given rapidly, 15 to 20 cc. per minute if the systolic pressure is below 90 mm. of mercury. This should be given within the first two hours. Further administration is given freely while the specific gravity (of whole blood) remains above 1.055, while the hematocrit values remain above 50 per cent, and the systolic blood pressure remains below 90 mm. mercury.

As soon as the patient's condition permits, broth, ginger ale, and later carbohydrate-containing fluids, strained cereals, gruel, sweetened tea, and fruit juices may be given.

Accurate records of fluid intake and output are essential.

IV. Secure an electrocardiogram as early as is practicable and repeat every four hours for twenty-four hours. More frequent tracings are indicated to guide therapy in cases of hypopotassemia. Careful evaluation of the electrocardiogram is considered by Nadler and his co-workers to be a much simpler method of detecting a hypopotassemia than quantitative analysis of the serum potassium. It is difficult to get a quantitative potassium analysis under six hours; whereas an electrocardiographic tracing can be obtained immediately.

V. Carbohydrates:

One should always keep hypoglycemia in mind when giving large doses of insulin. Careful observation by the physician, not by the nurse, and repeated urinalysis and blood chemistry are the only means of assurance that the patient is progressing satisfactorily. Therefore, it is advisable to give 1000 cc. of 5 per cent glucose four hours after therapy has begun. This should be repeated after six hours if the patient is not taking nourishment by mouth.

VI. Alkalies:

The administration of alkalies has been controversial. I do not believe that a patient with severe diabetic acidosis can be harmed by the administration of sodium R-Lactate solution. I do not hesitate to give these patients 600 cc. of 1/6 molar solution.

VIII. Potassium:

Because of the role of potassium in the treatment of diabetic coma and of the ill effects of hypopotassemia, I think one

should familiarize himself with the clinical physiology of potassium. The average concentration of serum potassium in the normal blood is from 4.0 to 5.4 milli-equivalents per liter or 16 to 21 milligrams per 100 cc. blood. The intra-cellular potassium is about 23 times that of the serum. During the development of acidosis much of the fixed base as well as potassium is lost.

During the treatment of diabetic coma, the extracellular fluid is expanded and the serum potassium level falls. Urinary excretion produces further loss of potassium. This continues until the potassium falls to a level producing respiratory paralysis and cardiac damage. That is why a number of patients in deep diabetic coma have improved at first then suddenly became worse and died. This catastrophe can be avoided by the judicious use of potassium when indicated. Potassium chloride should be given orally or by stomach tube, 1 to 2 grams at a time and repeated every four hours for 4 doses. For intravenous use, 100 to 500 cc. of a 1.14 per cent potassium chloride are administered. One must remember that it should not be given with poorly functioning kidneys.

VIII. Gastric Lavage and Enema:

The stomach should be emptied of its contents in case of abdominal distention, abdominal pain, or vomiting and 8 ounces of warm normal saline solution are left in the stomach. An enema is indicated in nearly every case of coma. It may be delayed until improvement in the patient's condition is noted.

IX. Diet:

When evidence of acute ketosis has subsided, a liquid diet is followed for twelve to twenty-four hours, and thereafter a diabetic diet of the same values totaling, for example: protein, 110 grams; fat, 65 grams; and carbohydrates, 250 grams or 2000 calories. The diet is given in four equal feedings, one every six hours; and finally, when complete recovery from the attack, the diet and insulin regimes as for the uncomplicated diabetic patients are resumed.

CONCLUSION

In closing may I state that there are several important factors in the treatment of diabetic coma: (1) Early recognition; (2) early administration of insulin in large doses; (3) early correction of blood chemistry by means of sufficient fluids and alkalis; (4) a careful watch for hypopotassemia, and hypoglycemia; (5) return of patient to oral medication as soon as possible; (6) careful search for the condition that precipitated the coma.

REFERENCES

1. Bertolini, A. M. and Scapellato, L.: Hypopotassemia in diabetic patients in coma, Giornale di Clinica Medica, Parma, 32:173 (Feb.) 1951.

2. Duncan, Garfield G.; Carey, Lawrence S., and Hudson, Manning T.: Diabetic coma, M. Clin. N. Am., Nov. 1949, p. 1537.

3. Fazekas, J. F. and Parrish, A. E.: Physiologic basis for treatment of diabetic coma, Med. Ann. Dist. Columbia, 18:171 (April) 1949.

4. Fisher, P.: Ketone bodies in etiology of diabetic coma, Am. J. M. Sci., 221:384 (April) 1951.

5. Guest, Geo. M.: Diabetic Coma, Am. J. Med., (Nov.) 1949, Vol. VII, No. 5.

6. Hoffman, William S.: Clinical physiology of potassium, J. A. M. A., (Dec. 2) 1950, Vol. 144, Number 14.

7. Joslin, Elliott P.: Futherance of treatment of diabetes mellitus, J. A. M. A., 139:4, (Jan.) 1949.

8. Joslin, Elliott P.: Treatment of diabetes today, J. A. M. A., 140:581, (June) 1949.

9. Lilly, Eli & Co.: Diabetes Mellitus, 1952.

10. Nadler, C. S., Bellet, S., Gazes, P. C.: Administration of potassium to patients with prolonged vomiting and diabetic acidosis, J. Lab. & Clin. Med., 35:842, (June) 1950.

11. Sinden, R. H., Tullis, J. L. and Root, H. F.: Serum potassium levels in diabetic coma, New England J. Med., 240:491, (March 31) 1949.

Spoont, Stanley, and Dyer, W. Wallace: Insulin resistance associated with local and general allergy to insulin, J. A. M. A., 145:558, (Feb.) 1951.

13. Sprague, Randall G.: Diabetic acidosis and coma, M. Clin. N. Am., (March) 1947, p. 457.

14. Stephens, F. Irby: Paralysis due to serum potassium during treatment of diabetic acidosis, Ann. Int. Med., Vol. 30, No. 6, (June) 1949.

15. Story, Robert D., and Root, Howard F.: Diabetic coma, J. A. M. A., (Sept.) 1950, Vol. 144, No. 2.

DIABETICS IN PREGNANCY*

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Diabetes is a serious complication of pregnancy in that the fetal mortality is much higher than in normal pregnancy. Before 1940, fetal mortality rates ranged from 30 per cent to 50 per cent. However, in the past decade, the growing awareness and combined efforts of the internist, pediatrician and obstetrician have been re-

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warded with a much lower fetal mortality rate. In fact, so much so, that today the physician may conscientiously assure the average diabetic that pregnancy may be undertaken with a good chance of delivering a living healthy infant.

General experience has shown that the diabetic of long standing (juvenile) with severe and diffuse vascular disease, hypertension, retinopathy and albuminuria has a much poorer chance of obtaining a living infant than a mild diabetic of short duration. However, regardless of severity certain complications occur with alarming frequency. These are:

1. Keto-acidosis—often with or secondary to upper respiratory infection or urinary tract infection.
2. Pre-eclampsia.
3. Progression of pre-existing hypertension with albuminuria and retinopathy.
4. Excessive edema with hydramnios.
5. Obstetrical complications, due to large babies, poor labor, and high incidence of breech presentation.
6. Physiological immaturity of infant, in spite of excessive size. These complications may result in fetal death at three periods:

1. (a) intrauterine death after thirty-two weeks' association with a maternal toxemia; (b) progressive hypertension, renal failure, etc.; (c) edema, hydramnia and hypertension; (d) keto-acidosis.
2. Intrapartum deaths due to accidents associated with delivery of extra large babies, stuck shoulders, and excessive traction.
3. Neonatal deaths due to (a) traumatic delivery, (b) prematurity, (c) and physiological immaturity of infants of diabetic mothers. Prenatal care should be undertaken only with the full realization that constant weekly supervision by both obstetrician and internist is the only way any degree of success can be attained. Successful control is based on:

a. Freedom from symptoms of diabetes.
b. Maintenance or average weight gain.
c. Freedom from keto-acidosis.

At each visit a complete urinalysis for sugar, acetone, and albumin should be done. In addition to the usual blood pressure and weight recordings, the abdomen should be palpated for size, position, and presentation, and the fetal heart tone checked on each visit. Eye grounds should be examined every week for evidence of vascular disease. Frequent blood chemistries, at least every two weeks up to thirty-two weeks and then every week, should be done. Because of the tendency to retain fluid even in the absence of true toxemia of pregnancy, patients should be given ammonium chloride daily with salt restriction after the sixteenth week.

Routine hospital admission at the thirty-fifth week for careful evaluation as to time and mode of delivery is advised.

The treatment of the complicated diabetic concerns itself with the management of the four previously mentioned conditions predisposing to intrauterine death, primarily after the thirty-second week.

The pre-eclamptic should be hospitalized immediately regardless of length of gestation, and pregnancy terminated if the infant is considered viable. There should be no hesitancy to terminate gestation as early as the thirty-fourth week if toxemia exists.

In the diabetic with established hypertension, probably the best criteria for progression are retinal changes. However, increases in blood pressure, albuminuria and nitrogen retention are indications for premature termination of pregnancy.

Those patients who develop excessive edema and hydramnios, even in the absence of hypertension, should be hospitalized and x-rays of the abdomen taken in an attempt to demonstrate fetal edema as represented by an area of translucency about the head and small parts, commonly referred to as a "halo". Ammonium chloride and mercurial diuretics are given to reduce edema of the mothers, but have little effect on the infant. Therefore, premature termination of pregnancy should be effected as soon as possible.

Keto-acidosis is prone to be associated with or follow upper respiratory infection or pyelitis, and is also noted more frequently

in those patients whose insulin requirements suddenly increase. These patients should be hospitalized immediately, seen in consultation with the internist, and measures instituted to control the acidosis. Pregnancy should be terminated as soon as possible after the condition is controlled.

In making the decision to terminate pregnancy prematurely in the diabetic, the obstetrician must weigh the hazards of prematurity against the dangers of intrauterine death.

Termination of gestation should be by cesarian section, or induction of labor, if conditions exist that would indicate a short easy labor could be anticipated. The shorter the duration of pregnancy the more likely a cesarean section will be needed. However, in a multipara with a soft, dilatable cervix an attempt at induction of labor should be made.

The excessive sized infant of diabetic mothers is an obstetric problem. There is a tendency to underestimate fetal size due to hydramnios and or edema of the abdominal wall. It is wise to use x-ray as an adjunct in determining fetal size as well as for fetopelvic disproportion. Cesarean section should be done, preferably under local or spinal anesthesia, in all cases in which excessive fetal size exists, or when labor is prolonged, or difficult delivery is anticipated. The employment of cesarean section under these circumstances will, of course, tend to lower the incidence of intrapartum fetal death.

Since there is a high incidence of primary atelectasis, followed often by pneumonia, the immediate neonatal period should be attended with tracheal catheterization, repeated at intervals to remove mucus and amniotic fluid. This is attendant with the administration of oxygen and heat control in an incubator. Further neonatal care is administered by the pediatrician.

A discussion of diabetes in pregnancy would not be complete without mention of the controversial question of hormone replacement therapy. The use of estrogen and progesterone as advanced by Priscilla White is predicated on the basis that their use will correct low serum estrogen and low pregnandiol excretion as well as high gonadotropic level in diabetic women.

Other workers do not use hormones, stating that it is difficult to see how such therapy could prevent intrapartum death due to trauma. Although White's reported fetal mortality rate is a low 10 per cent, no other group of workers has been able to approach a figure this low with or without hormone therapy.

In conclusion, it should be remembered that there is no single therapeutic agent which can be used to substantially lower the fetal mortality. Meticulous care, and frequent consultation between obstetrician, internist, and pediatrician are essential.

DIABETES IN CHILDHOOD*

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Holt and McIntosh's[1] textbook of pediatrics, published in 1939, opens its discussion of diabetes mellitus with the statement: "Diabetes mellitus is an uncommon disease of childhood." Subsequent experience is indicating with increasing evidence the inaccuracy of this statement. Diabetes can and does become overt at any age in life, from infancy to senility. And, either through greater awareness of this fact or through the increasing tendency on the part of physicians to include urinalyses as part of routine examination, more and more diabetics are being discovered during their pediatric years. It is currently estimated that from 5 to 8 per cent of all diabetic patients are children.[2] This is of particular importance in that it brings the patient to medical attention before the attrition of long-standing uncontrolled, or inadequately controlled, disease has made its inroads. The incidence of childhood diabetes approaches its peak as the child approaches adolescence. Five per cent of all cases occur in the first decade of life and about 12 per cent before the age of twenty years.[3]

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From the viewpoint of the geneticist, it is interesting to note that the disease may be diagnosed in the child before becoming apparent in the parent or older sibling.

MODE OF ONSET AND COURSE

With respect to pathologic physiology, there are no essential differences between the disease as it appears in adulthood and in childhood. However, there are variations in its mode of onset and its course which pose particular problems during the early years. Briefly and generally speaking, diabetes of children seems to be more abrupt in onset, more severe in its nature, and more erratic in its course. This is probably due more to the child than to the disease itself, and reflects the fact that during these years metabolic activity is at its peak. The anabolic processes of growth and development, as well as the extreme physical activity of the growing child, create a high energy demand which taxes to the utmost the integrity of the systems involved in digestion, absorption, conversion, and utilization of foodstuffs and, in doing so, tend to aggravate greatly any endocrine imbalance that may exist. It is all too true that anything that subjects the diabetic child to unusual stress, either physical or emotional, will be reflected in an increase in the severity of his disease. For this reason, children often first come to the attention of their physicians in ketosis, acidosis, or even coma. The premonitory signs of polydipsia, polyuria, and lassitude are mild and apt to go unnoted. The obesity which so classically brings the disease to mind in adulthood is characteristically lacking. The child may very likely be considered a behaviour problem because of his rather sudden change from a happy, energetic individual to a lethargic and irritable one. This idea may be further enhanced if, as is likely, there is a regression to nocturnal eneuresis in a child who had previously achieved efficient sphincter control. In this latent stage of the disease, the patient develops an upper respiratory episode or a similar mild infection which previously he would have been able to handle without difficulty, and his diabetes becomes rampant, resulting in the rapid onset of acidosis and coma and bringing him to medical attention as an emergency. It has been estimated that approximately 10 per cent of diabetic children are first seen in a state of coma. Because of this fact and the brevity and mildness of the prodromal period, physicians should be constantly on the alert for the existence of diabetes. Urinalysis should be a routine procedure, and any child who is brought to the office because of lassitude, failure to gain weight, developmental arrest or retardation, frequent recurrence of minor infections (particularly those of the skin such as impetigo, pyodermia, or furunculosis), or a return of nocturnal eneuresis should be thoroughly investigated with the possibility of diabetes in mind.

TREATMENT

In most instances the treatment of childhood diabetes involves two distinct phases: (1) the initial treatment of acidosis and electrolyte imbalance; (2) the long-term management of the child. Restoration of hydration and repair of the extracellular and intracellular spaces must be accomplished immediately. Adequate attention to the potassium deficiency, which is almost inevitable, is mandatory. During this phase, most practitioners will find the use of crystalline insulin most efficacious, though some workers claim advantages for the initial stabilization on protamine zinc or globin insulin.

The adequacy of long-term treatment of childhood diabetes will depend primarily on close cooperation among the parents, the physician, and the child. It has been said that the prognosis of diabetes is directly proportional to the willingness of the patient to learn all there is to know about the disease. During the early years the physician will be handicapped by the lack of mature judgment on the part of the patient, but this handicap will gradually diminish as the patient grows older. Early efforts at control must be rather exacting and discipline rather rigid; check-ups for the purpose of evaluation must be frequent. Later, as the patient becomes able to assume more and more responsibility, the trips to the

doctor's office and to the hospital may become less and less frequent. Long-term management must be fourfold in its objectives. In addition to the maintenance of chemostasis to as high a degree as possible, it must be concerned with the maintenance of normal growth and development, the avoidance or deceleration of degenerative changes, and the fostering of emotional health.

Initial regulation, even of the milder cases, must of necessity be a hospital procedure, and will be accomplished by determination of proper insulin dosage based on twenty-four-hour quantitative urinary sugar values. Refinement of dosage, spacing of injections, and the choice of insulin preparations will be dictated by fractional urine studies throughout the twenty-four hours. In the hospital and at home with children under the age of three years, diet can be controlled with relative accuracy. Above the age of three, this control will be difficult, following discharge from the hospital. For this reason, considerable support has been developed for control on an unrestricted diet. The wisdom of this is not entirely apparent and may not stand the test of time. Recent studies indicate that of the two factors involved in degenerative changes, i.e., adequacy of control and duration of disease, the former is the dominant. Therefore, it would seem that the establishment of a favorable dietary regime, though difficult to maintain, would be well worth the effort. The recognition of the difficulty in establishing a rigid dietary regime with children should prompt the physician to be as liberal as possible without sacrificing adequate control. The diet should contain all of the essential substances and adequate allowances for growth and activity. Most of the basic diets will adhere to the formula of 1.5 grams protein per pound of body weight per day for children under three years of age and 1 gram protein per pound for children over three years of age.[3] The remainder of the daily caloric requirement is equally divided between carbohydrates and fat. It is to be remembered that the onset of febrile illness increases sharply the daily caloric requirement and causes a temporary deterioration in the degree of glucose tolerance. For this reason every effort should be made to support the general health of the patient and cut down on the frequency of infection. The slightest infection, when it does occur, should be the signal for immediate and competent medical attention.

One of the less dramatic but nonetheless important aspects of long-term treatment is concerned with the emotional health of the child. Parents are extremely anxious and fearful and need constant reassurance. They are apt to be overprotective and solicitous, which is in its way just as detrimental to the child's developing personality as would be neglect. The patient himself does not want to feel that he is a "special case" and different from his playmates. He resents the rigid control to which he is subjected and may react violently with unacceptable symptomatic behaviour. These problems can and should be successfully met with patience and understanding on the physician's part. The diabetic child with proper care can lead a normal life and enjoy the pleasures of childhood to which he is entitled. He should not be kept from attendance at gatherings where occasional breaks in control are to be expected, such as birthday parties, Easter-egg hunts, and picnics. These activities, though perhaps temporarily detrimental to his diabetes, are very important to him as a person and contribute greatly to his emotional maturation and socialization. Athletics are not barred to him, nor is any activity that other young people enjoy. He must learn to accept his disease and live with it. Modern industry is finding that diabetes does not prevent full utilization of these patients, even under the unsatisfactory situation of shift-work. It is this adjustment of the patient to his diabetes for which the pediatrician must lay the ground work.

For those relatively few who can afford it, there are throughout the country a number of well managed summer camps for diabetic children which offer the child an opportunity for emotional growth and so-

cialization in addition to giving the parents a welcome respite from responsibility of their every-day care.[4]

NEWBORN OF DIABETIC MOTHERS

No discussion of childhood diabetes would be complete without mention of a problem that is the joint responsibilty of the obstetrician and pediatrician; that is, the newborn of diabetic mothers. Neonatal mortality is high, varying from 12 to 20 per cent in series in which maternal prenatal care was considered adequate to 60 to 70 per cent in series in which the care was not adequate. The incidence of congenital anomalies among these children is greater than in the general population. The explanation for this difficulty is not satisfactory. The babies tend to be large and postmature and, consequently, have a more difficult and dangerous journey down the birth canal. They have an unusal amount of respiratory distress with dyspnea and cyanosis for which there is no adequate explanation. There is an excessive amount of hemopoetic tissue and erythroblastosis is present often. Because of the frequency of convulsions and unexplained muscular twitchings, they were for a long time thought to be hypoglycemic. But recent studies, controlled by newborns of nondiabetic mothers, reveal that their blood sugar level is not excessively low.

At present, in view of our inadequate knowledge of the subject, the best we can offer is very careful observation of the newborn and a state of readiness to meet any difficulty which might arise. All deliveries should take place within a hospital; oxygen should be available; and tracheal suction should be resorted to if excessive mucous tends to occlude the airway. Glucose solution is frequently used if hypoglycemia is suspected.

REFERENCES

1. Holt, Emmett L., Jr. and McIntosh, Rustin: Holt's Diseases of Infancy and Childhood. D. Appleton-Century Company, Incorporated, New York and London. 1949.

2. Nelson, Waldo E.: Textbook of Pediatrics. Fifth Edition. W. B. Saunders and Company, Philadelphia and London. 1950, p. 1420.

3. Boyd, Julian D.: Practice of Pediatrics. Brennemann, W. F. Prior and Company, Hagerstown, Maryland. 1941. Vol. 3, chap. 12.

4. White, Priscilla and Waskow, Eleanor A.: Summer camps for diabetic children, M. Clin. North America. March, 1947.

5. Dunham, Ethel C.: Premature Infants: A Manual for Physicians. Children's Bureau Publication No. 325. U. S. Government Printing Office, Washington, D. C.

SURGERY IN DIABETES*

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As the years pass by, we of the surgical profession, find that surgery in diabetic patients is definitely on the increase. For this apparent increase it is felt that three reasons contribute largely to the more frequent use of surgery in the diabetic patient.

First, the lives of patients suffering from diabetes have been prolonged as a result of the more efficient manner in which the disease has been appraised and treated; so that many more diabetic patients are living to the ages in which surgical conditions develop or become acquired. Second, the incidence of diabetes, especially in patients above forty years of age seems to be definitely on the increase. Third, many improvements in surgical technique, preoperative and postoperative care, establishment of blood banks, and better anesthetic methods make it possible for many more elective procedures to be safely carried out in diabetic patients than was formerly possible.

As an illustration of this fact, as far back as the year 1947, the Mayo Clinic reported 477 surgical procedures carried out upon diabetic patients of all ages, with a hospital death rate of 12. This is a calculated mortality of 2.5 per cent. These surgical procedures comprised approximately 50 per cent of the major and 50 per cent of the minor type. This would indicate that surgery in the diabetic patient is not prohibitive and is well within the realm of reasonable safety. Diabetes can no longer be considered grounds for denying a patient the benefits of surgery and the decision to operate can now be based upon essentially the same criteria as among nondiabetic patients. However, the potential danger of

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surgical intervention in the presence of diabetes is as great as ever. The occurrence of acidosis and coma is still a possibility that can be prevented by close cooperation of the surgeon, internist, anesthetist, dietitian, and nursing staff.

COMPLICATIONS AND SEQUELAE

There are some common complications and sequelae that may follow surgery on the diabetic patient. In addition to the diabetic's carbohydrate intolerance he is further handicapped by the presence of arteriosclerosis, principally of the coronary and renal arteries. He also has a greater susceptibility to infection both in the operative wound and in the urinary tract. We must also consider the effect that acute or prolonged sepsis has upon the diabetic state, and also postoperatively, the frequency with which pressure, even of such degree as is of little consequence to the nondiabetic, produces decubital ulcers, and areas of necrosis particularly over the heels and the sacrum.

Infections, furuncles and carbuncles are probably the most common of diabetic complications that face the surgeon of today. These infections contributed largely to the mortality and the morbidity of the disease; however, since the advent of the antibiotics the prognosis of these conditions has been improved to a large degree. In the treatment of these conditions, adequate incision and drainage are still a sine qua non to be used in conjunction with adequate antibiotic therapy, covering the spectral range of organisms involved in the infectious process. An alternate procedure of aspiration of the pus cavity with instillation of penicillin has not gained general favor. In all possible circumstances, the diabetes should be adequately controlled to a satisfactory degree before any surgery is done.

Next in importance is gangrene, a common and frequent disabling complication of diabetes, and which constitutes a serious threat to the life of the elderly patient. In diabetic gangrene we recognize two factors as primarily responsible for the disability: First, there is present an ischemia, with a more or less advanced arteriosclerosis; anoxia and diminution in the effective circulation. Second, infection begins and progresses by the introduction of infection through abrasions or minor wounds of the skin with subsequent thrombosis in already diseased vessels. These two factors may combine to make gangrene unamenable to any form of treatment except radical surgical intervention. It has been stated that there is usually some degree of arteriosclerosis present, and this arterial insufficiency may be associated with cellulitis, lymphangitis, infection, or uncontrollable pain, so that amputation at a level where the circulation is adequate is inescapable. Since antibiotics have come into general use, in such conditions as cellulitis, lymphangitis, or infections, other than the gas producing ones, amputation may be delayed until these infections are greatly improved, by an effort to stabilize the diabetic state, with intensive antibiotic therapy, and dietary control. Likewise, if the gangrene is extensive there is little to be gained by procrastination. The two most essential features of the preoperative treatment of infected extremities are rigid control of the diabetes, and extensive antibiotic therapy. If the diabetes is not under control the amputation should usually be delayed until this has been achieved.

To illustrate the present day mortality in diabetic gangrene, I would like to quote the results of the Montefiore Hospital in New York City, 1950. In 196 patients, 213 midleg amputations were done. Of these 172 were done for diabetic patients with a mortality rate of 9.7 per cent; and 41 were done for arteriosclerosis obliterans with a mortality of 9.7 per cent. Reamputation was necessary in 4.7 per cent of the cases.

Lumbar sympathetic block in incipient gangrene, by the use of 1 or 2 per cent procain injected paravertebrally into the area of the sympathetic ganglia and chain has been used in an effort to increase the efficiency of the circulation and obviate the vasospasm that is present. Lumbar sympathectomy may be used and is a surgical block of the sympathetic pathway to the lower limbs. Some gratifying results have

been achieved by these methods, by delaying the immediate need for amputation, or by allowing amputation at a lower level than was previously anticipated.

Ulcers and other sores that constitute lesions of the feet that are not accompanied by any gross impairment of the circulation are caused as a rule by trauma and infection. They include infected corns, callouses, ulcers, burns, frostbite, varicose ulcers and abrasions. The diabetic patient should learn to consider all abrasions seriously, and to stay off his feet and meticulously attend to the cleanliness and care of them.

The ischemic ulcers that result mainly from local arterial insufficiency are classified into two groups. First, arteriosclerotic or diabetic ulcers, and second, decubitus ulcers. The localized areas of necrosis of the skin which occur on the extremities of patients with arteriosclerosis or diabetes are usually the result of anoxia following obliteration of the arterioles by thrombosis. From the standpoint of cellular pathology there is no difference between the ischemic ulcer of arteriosclerosis and diabetes. Uncontrolled diabetes, however, favors the spread of infection in such poorly nourished tissues. Physical, chemical, or thermal trauma to the skin may precipitate arteriolar thrombosis with subsequent ulceration of the skin. Decubitus ulcers or bed sores, are the result of abnormal sustained pressure over the bony prominences. They usually occur in patients whose general state of health and nutrition is below normal. General supportive and preventive measures are of paramount importance in dealing with this type of lesion. Surgical excision of these necrotic areas together with primary skin graft has been done with good results, provided that the diabetic state and infection have been adequately controlled. General supportive measures postoperatively, such as frequent change in position, and early ambulation constitute formidable preventive measures against decubitus ulcers.

CONCLUSIONS

In a general way, in considering surgery in the diabetic patient, we may conclude:

1. It seems to be definitely on the increase in occurence, and is probably due to the factors mentioned.

2. A determined effort should be made to stabilize the diabetic state, to correct the fluid and electrolyte balance, combat anemia and hypoproteinemia, if present, and to replenish the blood volume before elective surgery is done.

3. In acute abdominal conditions this evaluation may not be possible due to existing circumstances, and in these cases it seems wiser to operate and treat the diabetes later.

4. Lumbar sympathetic block with procain and lumbar sympathectomy are methods of salvaging in some instances a part of a limb that would otherwise be sacrificed, and are worthy of consideration.

5. In general, it may be stated that though the mortality rate for surgery remains higher in the diabetic than in the nondiabetic for comparable conditions, surgery is by no means a prohibitive venture and can be suggested to the patient with a reasonable assurance of safety, provided all of the preoperative requisites are satisfactorily met, and the postoperative therapy is adequate.

6. The patient should be followed by the internist both preoperatively and postoperatively, and therapy of the diabetic state in the convalescent and subsequent periods should be maintained by the internist.

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SOME MEDICAL COMPLICATIONS OF DIABETIC ACIDOSIS*

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PERIPHERAL VASCULAR COLLAPSE

Peripheral vascular collapse is a rather common complication of severe diabetic acidosis. It is primarily due to dehydration and to vasodilatation produced by changes in blood chemistry. Since this complication materially worsens the prognosis it is important to correct it early. Rapid and sus-

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tained elevations of the blood pressure may often be brought about by the judicious use of vasopressor agents such as l-norepinephrine. Permanent correction may require, in addition to the usual therapy with normal saline, plasma or whole blood.

MYOCARDIAL INFARCTION

Myocardial infarction occurs not infrequently. The diabetic state predisposes to early coronary sclerosis; peripheral vascular collapse further diminishes the coronary flow; and hypoglycemia due to overtreatment, of itself, may lead to this complication. In this connection, it is well to emphasize the great value of obtaining at least one electrocardiogram in every patient in coma, as well as to suggest the value of oxygen therapy throughout the coma period. Treatment, under the circumstances, involves meticulous control of anticoagulant therapy—which indeed may even be contraindicated—because of frequently coexisting hepatic or renal disease. In addition, the aglycosuric state should be avoided. Finally, it is necessary to use opiates and barbiturates only with great discretion.

CEREBRAL VASCULAR ACCIDENTS

Cerebral vascular accidents, for similar reasons, may complicate diabetic acidosis. Thus, it is important to repeat the neurological examination at intervals, and at times to repeat the lumbar puncture in comatose patients. These procedures have special value inasmuch as a developing meningitis might have been the precipitating cause of the acidosis, and again because occasionally a simple cerebral apoplexy may mimic diabetic acidosis.

CONGESTIVE HEART FAILURE

Congestive heart failure may occur against a background of preexisting heart disease, when the mycocardial reserve is depressed further by dislocation of electrolytes and nutrients, and it may be precipitated by myocardial infarction, or excessive administration of parenteral fluids, or both. In this situation the effects of digitalis are somewhat less predictable than usual. Also, the potassium therapy which is so widely used is fraught with added hazard since it has been reported that in the presence of heart failure the renal clearance of potassium is markedly reduced even when the urinary volume is normal.[1]

RENAL COMPLICATIONS

Among the renal complications of diabetes the Kimmelstiel-Wilson syndrome is of interest. It is characterized by hypertension and marked albuminuria occurring in relatively mild diabetes of long standing; renal function may be grossly impaired and nephrotic syndrome may occur. Arteriosclerotic renal disease is, of course, common in diabetes. The presence of impaired renal function greatly worsens the prognosis in diabetic acidosis. Such impairment may be due also to chronic pyelonephritis, and acute episodes of this condition may in turn precipitate, or complicate diabetic acidosis. The advantages of early and repeated microscopic examination of the urine—in contrast to following only the chemical tests—are therefore apparent. Functional renal impairment with oliguria and azotemia may arise in consequence of hypovolemia and peripheral vascular collapse, and is correctible by restoration of the circulation. Acidosis *per se* tends to diminish the kidneys' ability to conserve base, thus furthering progression of the acidosis. This situation may require the administration of alkali, such as sodium bicarbonate.

ALTERATION OF POTASSIUM METABOLISM

Alteration of potassium metabolism in diabetic acidosis is now widely recognized as a factor of great clinical significance.[2] Early in the development of acidosis potassium is lost from the cells into the extracellular fluid, and, with the diuresis of hyperglycemia, is lost in the urine, thus tending to deplete the body stores of potassium, while the serum potassium concentration is not greatly altered. However, in the presence of renal impairment, and in those diabetics who become oliguric, the serum potassium may actually rise. A more frequent cause of hyperkalemia is the injection of excessive amounts of this ion in the course of treatment. Transient paresthesias, neurological changes, weakness of various muscle groups, have all been described as symp-

toms of potassium intoxication, but none of them consistently. Arrhythmias may be detected clinically; however, the electrocardiogram offers the surest guide to the presence of this syndrome. Changes described include high narrow T-waves; broad, slurring QRS complexes; absence of P-waves; irregular undulations of low voltage leading to cardiac arrest.[3-6] Of these, the high, narrow, T-wave is the most consistent and most definite change. The serum potassium determination, while useful when available, does not necessarily reflect the clinical status, since the clinical effect of potassium alterations is modified by the levels of other cations. Treatment of hyperkalemia is initiated by the use of insulin and glucose which drives potassium into the cells; sodium and calcium antagonize to some extent the effects of potassium; and supportive measures designed to restore urinary output facilitate the elimination of this ion. The artificial kidney is reported to be of great value in this syndrome,[3] but is unfortunately not generally available; however, continuous gastric lavage is capable of removing moderate amounts of potassium, and it might be tried should the situation appear to warrant a measure which is beset with the difficulty of controlling concomitant changes in other ion concentrations.

Hypokalemia, or low serum potassium, is more common in the course of diabetic acidosis. In the presence of low body stores of potassium, continued diuresis resulting from hyperglycemia, fostered by acidosis, and increased by rehydration, coupled with the hemodilution of rehydration and the reentry of potassium into the cells under the influence of glucose and insulin, all conspire to diminish markedly the level of circulating potassium. Clinical signs of hypokalemia include muscular weakness, frank paralysis, abdominal distention, cardiac dilatation, systolic murmurs, and cardiac arrhythmias. The electrocardiogram, again, is most useful in evaluating the net effect of the altered potassium levels. A great many changes have been described in hypokalemia.[6-9] Of these the most consistent is the finding of wide, rounded, low T-waves. The prolonged Q-T interval which is described is the result of the increased duration or width of the T-wave. (It is to be differentiated from the prolonged Q-T interval of hypocalcemia which is the result of increased duration of the S-T segment.)[7] The T-wave in hypokalemia may also be inverted, and it may be associated with a depressed S-T segment, and with prominent U-waves. Here again the serum potassium level may be helpful, but is not essential, nor does it consistently reflect the net effect of the patient's potassium status. This level may be normal despite severe depletion of the body potassium stores. The critical factor appears to be the effective concentration of the ion at the surface of the cell membrane. Treatment of hypokalemia consists in the administration of potassium salts in quantity sufficient to improve the patient, rather than to restore the serum level to theoretical normal. The oral route is to be preferred whenever possible, and one gram of potassium chloride at hourly intervals for four doses is often sufficient to tide the patient over until normal feedings—which is the best source of potassium—can be started. In emergency, it may be necessary at times to give potassium salts parenterally. Darrow's solution is generally available and may be used, diluted with an equal volume of glucose solution to yield a fluid containing 17.5 milliequivalents of potassium per liter. Whatever solution is used, the important thing is to give it slowly, and preferably to follow the patient with electrocardiograms throughout this critical period.

REFERENCES

1. Brown, Harold, Tanner, G. L., and Hecht H. H.: The effects of potassium salts in subjects with heart disease, J. Lab. & Clin. Med., 37:506, 1951.

2. Darrow, Daniel: Body fluid physiology: The role of potassium in clinical disturbances of body water and electrolytes, New England J. Med. 242:928 and 1014, 1950.

3. Merrill, J. P., Levine, H. D., Somerville, W., and Smith, S.: Clinical recognition and treatment of acute potassium intoxication, Ann. Int. Med., 33:797, 1950.

4. Keith, N. M., and Burchell, H. B.: Clinical intoxication with potassium: Its occurrence in severe renal insufficiency, Am. J. M. Sc., 217:1, 1949.

5. Wener, J., and de Leeuw, N. K. M.: Hyperpotassemia and electrocardiographic changes in uremia, Canad. M. A. J., 61:406, 1949.

6. Bellet, S.: Clinical syndrome of hyperpotassemia and

hypopotassemia, Pennsylvania M. J., 53:708, 1950.

7. Ernstene, A. C., and Proudfit, W. L.: Differentiation of changes in Q-T interval in hypocalcemia and hypopotassemia, Am. Heart J., 38:260, 1949.

8. Perelson, H. N., and Cosby, R. S.: Electrocardiogram in familial periodic paralysis, Am. Heart J., 37:1126, 1949.

9. Bellet, S., Steiger, W. A., Nadler, C. S., and Gazes, P. C.: Electrocardiographic patterns in hypopotassemia: Observations on 79 patients, Am. J. M. Sc., 219:542, 1950.

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TREATMENT OF FRACTURES OF THE HIP*

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SHREVEPORT

The term "broken hip" is commonly used to designate fractures of the femur in the region of the neck and the greater and lesser trochanters. Fractured "hips" have been variously classified in the past. We have classified such injuries into two major groups: from the standpoint of prognosis in healing, and also, from the standpoint of method of internal fixation. The two major groups thus designated are:

1. Intracapsular fractures of the neck of the femur, i.e., fractures through the narrow portion of the neck.

2. Trochanteric fractures, i.e., fractures involving the region of the greater and lesser trochanters.

The above injuries have been observed most frequently in individuals who are considered "elderly" by most of us. It is generally recognized that prolonged confinement to bed and inactivity in this age group hastens debilitation and often makes it impossible to restore the patient to activity that is normal for him.

HISTORY OF TREATMENT

In 1902, Royal Whitman, of the Hospital for Ruptured and Crippled, presented his new method of treatment of fractured hips. This consisted of reduction of the fracture site by abduction and internal rotation under anesthesia followed by the application of a long plaster spica extending from the toes on the affected side to the nipple line. He demonstrated on the cadaver and in the operating room that this method was adequate to obtain reduction. This method was designed especially for intracapsular fractures of the neck of the femur and constituted the first major advance in the treatment of this condition.

In 1927, Wikie described a well leg traction system. In 1932, Roger Anderson devised a well leg traction splint that held the affected extremity in internal rotation. In this method of treatment each lower extremity was incorporated in plaster and allowed the patient to sit up.

The evolution of the surgical treatment of fresh fractures of the hip is also interesting. In 1850, Langenbeck attempted to insert a nail through the trochanter and into the head. This was repeated by Konig in 1875, by Trendelenburg in 1878, and by Nicolayesen in 1897. Different types of nails were used. In 1907, Lambotte and Delbert used ordinary screws. In 1919, they reported one successful case. This was not sufficient to justify general use of the method. In 1911, Borelius used bone pegs as internal fixation. In 1912, Albee used a similar method. These methods were found to be ineffective, chiefly because of inadequate reduction and unsatisfactory placement of the nail, and also, because the nail or screw did not prevent rotation of the head on the nail or screw. In 1925 Smith-Petersen devised a triflanged nail that would prevent rotation of the head. The nail was inserted under direct vision by open reduction. This added to the success of internal fixation. In 1932, Johansson of Sweden and Westcott of Virginia, working independently, reported "blind nailing" using x-ray as a guide.

In 1934, Henderson of the United States and King of Australia independently cannulated the Smith-Petersen nail that was inserted following correct positioning of a guide wire. In 1937, Thornton devised a plate to be attached to the Smith-Petersen nail and extend along the femoral shaft. The latter was used in trochanteric fractures. Many modifications of this plate have since been devised.

In 1938, the most important of contribu-

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tions was made by Venable and Stuck. This followed their study of reaction of body tissues and fluids with metals. It was determined that metals that cause electrical potentials when applied to bone and covered with soft tissue cause necrosis and absorption of bone, and therefore, become loosened. An alloy that had recently been used in dental prosthesis was studied. The alloy was composed of cobalt, chromium, and molybdenum and had been given the name vitallium by the manufacturer. It was found that this alloy possessed the least electrolytic coefficient when in contact with human tissues. It therefore became established that the use of nonelectrolytic metals in all bone surgery was essential. Following this, 18-8 SMO stainless steel was developed and also found to be practically inert, and thus, suitable as internal fixation.

PREOPERATIVE MEASURES

In fractures of the hip, as in other surgical conditions, the preoperative condition of the patient must be considered. A complete blood count and urinalysis are obtained. If the patient is anemic, he is typed and cross matched for transfusions. As a rule, the older group of patients are anemic and need blood because of that. If the anemia is not too marked the blood is given at the time of operation. At times marked anemia demands that blood also be given prior to surgery. A urinalysis is done chiefly for the sake of determining if the patient has sugar in the urine. If so, a blood sugar is obtained and appropriate steps are taken by the internist prior to surgery. Other pathological findings in the urine should be treated postoperatively.

The question asked us most often by relatives of the patient is, "Is the patient's heart good enough to stand the operation?" Our opinion is that if the patient is ambulatory and does not show signs of congestive heart failure, his heart is good enough to carry on during surgery. We never minimize the risk of anesthesia or surgery, regardless of age, but impress upon those concerned that there is always a risk to all surgery. However, we believe that delay in surgery for more than one or two days after the injury is detrimental to the older age group of patients.

Another point that should be stressed is avoidance of over-sedation. Again, since this type of injury is more frequent in older people, it should be emphasized that oversedation slows the patient's heart and respiration so as to increase surgical risk sufficiently to justify postponement of surgery. This avoidable delay of surgery is detrimental to the patient.

METHOD OF TREATMENT

A brief outline of our method of treatment is as follows:

Preoperative sedation is slight or none at all. We prefer to use an anesthesiologist to give the anesthetic for our patients. The anesthesia used in practically all cases is sodium pentothal and nitrous oxide. Care is always exercised to keep the patient under "light" anesthesia. The hip is then manipulated and each lower extremity immobilized on the orthopedic table. Two portable x-ray machines are placed in position, one for the anteroposterior view and one for the lateral view of the hip. Having two x-ray machines and a developing room adjacent to the operating room is a very important factor in saving time in this operation. An anteroposterior and lateral x-ray is then taken to verify reduction of the fracture. By the time the films have been developed the surgeons have scrubbed and the operative site has been prepared. As a rule, an incision about 3 inches in length is made along the long axis of the thigh through the skin, deep fascia and underlying tensor fascia femoris at the level of the base of the greater trochanter. Several guide wires of equal length are at hand. One of these wires is drilled through the long axis of the neck of the femur into the head. The correct position of the guide wire is then verified by an AP and lateral x-ray of the neck of the femur. The correct length of the cannulated Smith-Petersen nail to be used is determined by comparing the inserted guide wire with the uninserted guide wire of equal length. The nail is then threaded over the wire and

TABLE 1

Hips	No.	Per Cent	Average Age	Male No.	Male Per Cent	Female No.	Female Per Cent
Intracapsular	100	47.4%	68 yrs.	13	13 %	87	87 %
Trochanteric	111	52.6%	73 yrs.	26	23.5%	85	76.5%
TOTAL	211	100 %	70.5 yrs.	39	18.4%	172	81.6%

driven into place. At this stage of the operation, if we are treating a trochanteric fracture, the skin incision must be prolonged and the vastus lateralis reflected from the femur to allow for attachment of the Thornton plate to the Smith-Petersen nail and the distal fragment of the femur. The reason for the delay in making an adequate incision, so as to be able to apply the Thornton plate, is to avoid undue bleeding. When the vastus lateralis is reflected, or its fibers separated, there are often multiple minute bleeding points that continue to ooze while the fibers are separated or the muscle retracted. The oozing promptly stops on return of the muscle to anatomical position and with slight pressure. Following hemostasis the wound is closed in layers. The patient should be waking up at the end of the operation. On return to the room, deep breathing is encouraged and the patient is turned frequently. The following day the patient is placed in a wheel chair. This is done once or twice daily. Quadriceps exercise is started on each lower extremity to prevent muscular weakness. Care must be taken to see that the patient voids and defecates normally. The patient usually returns to the care of the family physician by the end of one week. If the patient is strong enough he may walk with crutches or a walker without weight bearing. The fracture site is observed by x-ray every six weeks until healing takes place or in unfortunate cases, until it is ascertained that further surgery is indicated.

ANALYSIS OF CASES

In this study we have reviewed the cases treated beginning January 1946, until about the middle of 1951, a period of five and one-half years. We have been able to obtain adequate follow-up study on 211 hips. The method used in treating all of these cases is that of internal fixation. The cannulated Smith-Petersen nail was used in the intracapsular fractures and the Thornton plate was added to this in the trochanteric fractures.

Table 1 shows that 100 or 47.4 per cent of the 211 cases were intracapsular fractures. The average age of this group of patients was 68 years. Thirteen per cent of the cases were males and 87 per cent were females. The trochanteric fractures numbered 111, being 52.6 per cent, with the average age of 73 years. Of this group 23.5 per cent were males and 76.5 per cent were females. The average age of the 211 cases, or the entire group, was 70.5 years, these being 18.4 per cent males and 81.6 per cent females.

TABLE 2

FOLLOW-UP STUDY OF INTRACAPSULAR AND TROCHANTERIC

Period	Hips Number	Hips Per Cent
Less than 1 year	25	11.8
1 to 2 years	72	34.1
Over 2 years	91	43.1
Deaths	23	10.9
TOTAL	211	99.9

The period of follow-up study has varied from about eight months to over two years. This is shown on Table 2.

TABLE 3

MORTALITY — INTRACAPSULAR

Age	Number of Hips	Deaths Number Within 1 Month	Deaths Number Within 1 Year	Deaths Per Cent Within 1 Month	Deaths Per Cent Within 1 Year
Under 40	3	0	0	0	0
40 - 49	4	0	0	0	0
50 - 59	15	0	0	0	0
60 - 69	24	0	0	0	0
70 - 79	32	0	3	0	9.4%
80 and over	22	2	2	9.1%	9.1%
TOTAL	100	2	5	2.0%	5.0%

The intracapsular fractures will first be considered as a separate group of patients. In Table 3 the mortality as to age group is

given. It is noted that only 7 of the 100 cases is under 50 years of age and only 22 under 60 years of age. In the older group of cases 2 deaths occurred during the month following operation. The cause of death in one case was uremia and the cause in the other was coronary occlusion. Five additional deaths occurred in the two older age groups during the year following operation. Two of these occurred six weeks after operation and were due to cardiac failure. The remaining 3 died the latter part of the year, 1 of cerebral hemorrhage, 1 of cerebral arteriosclerosis and 1 of uremia. Considering only the 2 cases dying within the month following surgery, there is a mortality of 2 per cent. Considering the entire group dying during the year following surgery there is a mortality of 7 per cent.

Practically all patients were returned to the care of the family physician by the end of one week after operation. Therefore, it must be said that no doubt other deaths occurred in other cases not considered here that we were unable to follow.

Two of the cases dying in the latter part of the year following operation had union of the fracture site. The remaining 5 cases died before sufficient time elapsed for union to take place. This leaves a total of 95 cases to be considered in further study. Table 4 presents the number of hips and percentage of union and nonunion according to decades. It is noted that 90 cases or 94.7 per cent of the cases obtained union and 5 or 5.2 per cent of the cases went on to nonunion.

In the 100 cases of intracapsular fractures studied the nail slipped out of the head of the femur in 3 cases. Two of these cases refused further treatment and are included in the cases of nonunion.

TABLE 4

INTRACAPSULAR FRACTURES

	Union		Non-Union	
Age	No. of Hips	Per Cent	No. of Hips	Per Cent
Under 40	2	66.6	1	33.3
40 - 49	4	100	0	0
50 - 59	15	100	0	0
60 - 69	23	95.8	1	4.1
70 - 79	29	96.6	1	3.3
80 and over	17	89.4	2	10.5
TOTAL	90	94.7	5	5.2

In the group of 90 cases in which union occurred, 7 cases or 7.7 per cent developed moderate aseptic necrosis of the head of the femur. These patients complained of pain in the hip on motion. The condition was shown on x-ray examination as some increased density of the head with mild to moderate roughening of the articular surface of the head. It was noted in one case in the fifth decade, 3 cases in the sixth decade, and 3 cases in the eighth decade. The above symptoms and signs were usually noted by the end of one year. However, one case developed a painful hip and the roughening of the head of the femur was noted two years following surgery.

The incidence of painful hips following an intracapsular fracture is greater than the mortality or the incidence of nonunion, and in the future may offer an increasing problem.

TABLE 5

MORTALITY — TROCHANTERIC

		Deaths			
		Number		Per Cent	
Age	Number of Hips	Within 1 Month	Within 1 Year	Within 1 Month	Within 1 Year
Under 40	2	0	0	0	0
40 - 49	8	0	0	0	0
50 - 59	7	1	0	14.2	0
60 - 69	21	2	2	9.5	9.5
70 - 79	40	2	5	5.0	12.5
80 and over	33	0	4	0	12.1
TOTAL	111	5	11	4.5	10.0

The trochanteric fractures are presented in Table 5 as to age and number of hips, according to decades and the mortality occurring in each decade. It is noted that only 10 fractures, or 9.0 per cent, occurred in patients under 50 years of age. The one death in the sixth decade occurred because of pulmonary embolism two weeks following surgery. In the seventh decade, 2 cases died before the end of one month. One died on the day of the operation from shock and the second died fifteen days after the operation from pulmonary embolism. The re-

maining two deaths occurring in this decade were at the end of four months from thrombosis of the inferior vena cava and at the end of six months from diabetes. In the eighth decade 2 deaths occurred at the end of one month following surgery, 1 as the result of pneumonia, and the other as the result of uremia. The remaining 5 deaths occurred at the end of three to six months and resulted from cardiorenal failure, uremia, cerebral hemorrhage, and pneumonia. In the ninth decade no deaths occurred in the month following operation; however, four cases died during the following four months as the result of cardiac failure, debility, pneumonia, and cerebral hemorrhage.

It is interesting to note that of the 211 cases of broken hips presented, only 1 died of shock immediately following the operation. We believe that this probably could have been prevented, had better nursing care been available at the time.

In all cases of trochanteric fractures, bony union occurred. In no case did aseptic necrosis of the head of the femur take place.

REFERENCES

1. Bick, Edgar M.: Source Book of Orthopedics, Williams & Wilkins, Baltimore, 1948.

2. Cleveland, Mather and Bosworth, D. M.: Intertrochanteric fractures of the femur. A survey of treatment in traction and by internal fixation, J. Bone & Joint Surg. 29:1049 (Oct.) 1947.

3. Boyd, H. B. and George, I. L.: Complications of fractures of the neck of the femur, J. Bone & Joint Surg. 29:13 (Jan.) 1947.

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TOXIC REACTION FROM GOLD APPEARING DURING ACTH ADMINISTRATION*

REPORT OF A CASE

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The statement has appeared in recent months[1-3] that cortisone and corticotropin are superior to BAL in the treatment of toxic reactions resulting from the adminsistration of gold compounds in rheumatoid arthritis. The following is an interesting example of a severe gold reaction occurring while the patient was receiving ACTH simultaneously, and which cleared up following a course of treatment with BAL.

CASE REPORT

B. K., a 36 year old unmarried white male, was first seen at the Browne-McHardy Clinic on August 31, 1950. The chief complaint was that of bilateral shoulder pain of a dull, aching type, appearing about ten days previously. Three days later there was an increase in the shoulder pain on awakening, accompanied by swelling and pain in the third, fourth, and fifth metacarpal phalangeal joints of both hands. A physician was consulted and it was suggested that the symptoms were on an allergic basis. Treatment consisted of the administration of sodium salicylate and pyribenzamine, with gradual disappearance of his complaints. Fifteen years previously there had been a nonspecific type of rheumatic difficulty in the right knee with no acute manifestations. This had apparently cleared up in a short time and left no residuum. An additional complaint when first seen by us was that of epigastric discomfort off and on for about two years. This was accompanied by acid regurgitation which was relieved by food.

The past history was essentially negative except for gonorrhea in 1934, and appendectomy for a ruptured appendix in 1932. A positive serology for syphilis appeared shortly after a severe reaction to an injection of tetanus antitoxin in 1935. The patient had been checked repeatedly by physicians in New York City and elsewhere without establishing the presence of a luetic infection. He denied having a primary or secondary lesion and the general conclusion reached was that the reaction was a "false positive" one.

Physical examination at the time he was first seen by us revealed him to be a well developed, well nourished, somewhat nervous white male. There were no positive joint findings at this time and the examination was otherwise essentially negative except for a small rectal polyp which was cauterized. When examined pathologically, malignant degeneration at the tip was reported.

Initial laboratory studies revealed a hemoglobin of 15.2 grams, red blood count 5,290,000, white blood count 11,600, with a differential of 76 neutrophiles, 1 eosinophile, 22 lymphocytes and 1 monocyte. The sedimentation rate was 45 mm./hr. by the Westergren method. Serologic tests were reported as follows: Kahn doubtful, Kolmer negative and Kline positive. X-ray of the chest revealed slight fibrosis at the right apex and calcified hilar nodes. Gastrointestinal series revealed a diffuse gastritis and slight irritability in the second portion of the duodenum.

Other than the rectal polyp and the gastritis, no positive diagnosis was established at this time, especially in the absence of objective joint manifestations. The gastrointestinal symptoms responded to a program of medical management. The

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patient was not seen again until January 20, 1951. He stated that he had begun to have aching and stiffness in the left wrist, elbow, and shoulder about ten days previously. This had cleared up but was followed by pain in the left knee and the left third metacarpal phalangeal joint, as well as by backache. Objectively, there was grade 2[1] swelling and tenderness of the left third metacarpal phalangeal joint. The joints were otherwise within normal limits objectively. The sedimentation rate, however, had increased to 75 mm./hr. and it was felt that the diagnosis at that time was early rheumatoid arthritis. He was advised to take aspirin and apply heat to the affected joints, and then was not seen again until April 11, 1951. He stated that he had continued to have pain and stiffness in both wrists and hands. His job had kept him traveling over parts of the country where he consulted numerous physicians and had two teeth extracted, as well as treatment directed toward correction of a pyorrheal condition without improvement of the basic process. He was referred at this time for arthritic consultation where the diagnosis of early rheumatoid arthritis with minimal fibrositic component was concurred in. Objectively, there was grade 2 synovitis and tenderness of the left wrist with considerable pain on movement. There was 1– synovitis of the left third metacarpal phalangeal joint and the grip was weak in both hands. There was no other involvement objectively. The sedimentation rate at that time was 59 mm/hr. A full conservative program of treatment was outlined. This included extra rest, both physical and mental, high caloric, high vitamin diet with daily multivitamin supplement, aspirin as needed, and physical therapy measures for the left wrist and hand including the use of contrast baths and muscle strengthening exercise within the limits of tolerance. X-ray of the left hand and wrist on May 2, 1951, showed soft tissue swelling about the interphalangeal joints and definite destructive arthritic changes in the carpal, scaphoid, and semilunar bones, especially about contiguous articular surfaces. There were small cystic areas near the articular cortex of some of the other carpal bones. The changes in the carpal bones were felt to be definitely rheumatoid in nature.

Cooperation on the part of the patient in treatment was not optimal and the rheumatoid process continued active. Despite a considerable degree of disability and his inability to carry on his work, he seemed unable to accept the diagnosis and offered this as an excuse for not carrying out recommended treatment measures. On July 10th, there was a flare-up in both knees for the first time and the left shoulder became quite stiff. He reacted with a considerable degree of panic, especially over possible implications regarding his ability to continue work. Consequently, it was decided to supplement the above program with a course of gold (gold sodium thiomalate or myochrysine). The sedimentation rate at that time was 68 mms./hr. by the Westergren method; there was moderate activity in the shoulders, elbows, and ankles, as well as in the wrists and hands.

Routine checks done prior to gold administration were within normal limits except for 5 per cent retention of bromosulphalein at the end of thirty minutes. At the time this was not believed to be of significance, especially in the absence of a history of previous liver disease and it was felt that there were no contraindications to gold therapy. The initial dose of myochrysine on July 13, 1951, was 10 mgms. followed by 25 mgms. the following week, and then a weekly maintenance dose of 50 mgms. A total of 435 mgms. was reached on September 7, at which time the drug was discontinued (Fig. 1).

On July 27, 1951, the patient developed a rather severe flare-up with pain and stiffness in the neck and temperomandidibular joints. For several days he was unable to open his jaw more than 1/4 inch. This cleared gradually but there was continued pain and stiffness in the shoulders and right elbow, as well as in the wrists and hands. The grip remained exceptionally weak and muscle strengthening exercises were quite painful. Because of his downhill course he was admitted to Touro Infirmary, New Orleans, Louisiana, on August 28, 1951, for a short course of ACTH in an effort to determine if this would supplement the gold effect. Dosage of myochrysine had reached 335 mgm. at that time. The ACTH[2] was administered as follows: 25 mgms. every six hours for five days, at which time dosage was gradually tapered off over a four day period. Total dosage was 800 mgm. There was excellent response within forty-eight hours and at time of discharge on September 7, 1951, there was only minimal (1–) tenderness over the head of the left ulna. No pain or stiffness was present in other joints and the hands and wrists had regained almost all of their normal strength. There had been no evidence of gold toxicity either from a clinical or laboratory standpoint until this time.

The patient returned to work the following day, stating that he felt better than he had in many months. He was sufficiently strong to embark on a road trip for his employer, the first such assignment in approximately five months. On September 11, he telephoned from Houston, Texas, and stated that his mouth had become quite sore and painful over a three day period, and on consulting a physi-

[1]Findings in the joints were graded on a basis of 1 to 4, 4 plus being maximal involvement.

[2]ACTHAR C—Supplied by Dr. Harley M. Cluxton, Director of Medical Research, the Armour Co., Chicago.

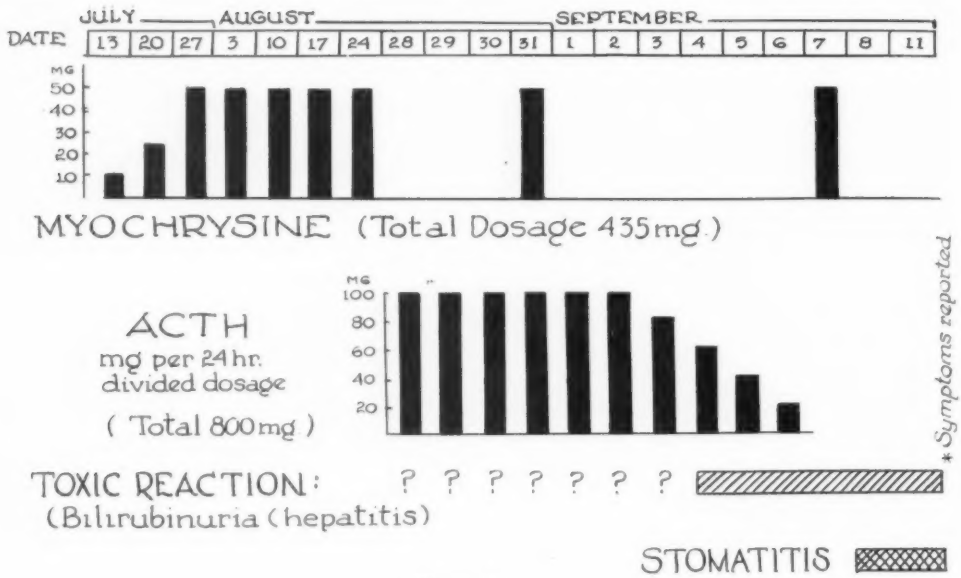

Figure 1.

cian there, he was found to be jaundiced. In retrospect, he reported that his urine had been quite dark for at least a week (*i.e.*, during the period while he was still receiving 9CTH) but he neglected to report this while in the hospital, thinking it of no significance. Routine urinalysis prior to the last injection of myochrysine on September 7, 1951, had been reported as negative, unfortunately. The presence of bile was apparently overlooked. He was advised to return to New Orleans where he was hospitalized immediately. On examination he stated that he felt nauseated, and that his hands shook continuously. His mouth was quite sore and examination revealed many small aphthous ulcers. He was obviously jaundiced and the liver edge was palpable but the spleen could not be felt. Laboratory work at that time was as follows:

On September 12, 1951, red blood cells 4,320,000 cu/mm; white blood cells 10,000 cu. mm.; neutrophils 70; eosinophils 3; lymphocytes 24; monocytes 4; blood platelets 190,000 cu/mm; urine—positive for bile; urobilinogen positive in dilution 1/160; serum bilirubin 7.95 mgm. per cent; total serum proteins 6.0 gm. per cent; albumin 4.2 gm. per cent; globulin 1.8 gm. per cent; thymol turbidity 7 units (normal 0-4); cephalin cholesterol flocculation 3 plus (normal 0-1).

September 14, 1951, prothrombin time 15.7 sec.—97 per cent.

September 18, 1951, serum bilirubin 6.3 mgm. per cent.

September 22, 1951, liver biopsy reported as follows: Bile thrombi within the mid and central portions of the lobules consistent with a cholangiolytic hepatitis due to gold therapy (Sections reviewed by Dr. A. J. Hertzog, Pathologist, Touro Infirmary, New Orleans).

September 24, 1951, serum bilirubin 4.9 mgm. per cent; alkaline phosphatase 11.1 Bodansky units; prothrombin time 16.1 sec. –87 per cent; total serum proteins 6.6 gm. per cent—albumin 4.5 gm. percent; globulin 2.1 gm per cent; thymol turbidity 7 units; cephalin cholesterol flocculation –4 plus.

September 28, 1951, serum bilirubin 3.1 mgm. per cent.

The second period of hospitalization extended from September 12, to October 5, 1951, during which time he developed a full-blown gold dermatitis of the exfoliative type as well as the hepatitis and stomatitis. After consultation with Dr. Howard Polley of the Mayo Clinic, it was felt that the toxic reaction should be treated with BAL, especially since the toxic manifestations had come on while the patient was receiving ACTH. The preparation of BAL used was one of 10 per cent BAL and 20 per cent benzylbenzoate in peanut oil given according to the schedule recommended by Eagle; namely, 3 mgms./kg. every four hours on the first and second day, every six hours on the third and fourth day, and then twice daily on the fifth and sixth days. The drug was discontinued at this time because of an extremely satisfactory response on the part of the patient. Specific therapy was supplemented by a high carbohydrate, high protein, low fat, liver type diet, daily glucose infusions with large doses of vitamin B and C added, a preparation containing the lipotropic fac-

tors including choline, methionine and isotol, and multivitamin supplements. The patient was kept at strict bed rest and likewise received symptomatic treatment to the mouth and skin as needed. He responded nicely to this program and was discharged from the hospital about October 5, 1951, at which time he returned to his home in New York City to convalesce. Interestingly enough, the joint symptoms remained in complete abeyance during this period and had not returned at the last time he was seen for a check-up on Jan. 25, 1952. Significant laboratory studies at this latter date were a serum billirubin of 0.9 mgms. per cent, bromsulphalein dye retention of 5 per cent at the end of thirty minutes (essentially the same as at the onset of the illness) and a thymol turbidity of 4 units. Urine was negative for bile; sedimentation rate was 41 mms./hr.

COMMENTS

Reference to the diagram will illustrate graphically the onset of the gold reaction during the administration of ACTH. It is interesting to speculate as to the possibility of latent liver damage in this patient which might have accounted for the probable false positive serology and bromsulphalein dye retention of 5 per cent which had been noted prior to the use of gold. Many authorities[1] feel that previous kidney or liver disease is not a contraindication to gold therapy unless they seriously impair the function of these organs. Even then the danger is not that toxicity is more apt to occur but rather that if severe toxicity from gold is encountered, the summation of difficulties may produce serious consequences.

It is obvious from the data that the gold reaction was first apparent at about the time the doses of ACTH were reduced with the idea of tapering it off gradually, although it may have been developing subclinically for some time before. Whether the reaction would have been prevented if ACTH had been continued in full dosage for a longer period of time is, of course, unknown. Since the reaction was not anticipated, however, and the main purpose of the first hospitalization had been an effort to determine the possible effectiveness of ACTH in supplementing gold effects, studies of adrenocortical function had not been done at that time. All of these factors led to the decision to use BAL in the treatment of the reaction.

SUMMARY

1. A case is presented in which a toxic reaction to a gold salt (myochrysine) apeared while the patient was receiving ACTH. The reaction consisted in the development of a hepatitis of the cholangiolytic type, stomatitis, and exfoliative dermatitis.

This reaction was treated with BAL and responded very well.

REFERENCES

1. Duff, I., and Robinson, W. D.: At University of Michigan Post-Graduate Course in Rheumatic Diseases, Ann Arbor, April 26, 1951.

2. Coste and Oury: Other uses of ACTH and cortisone, J.A.M.A., 145:1286 (April 21) 1951.

3. Steinberg, C. L., and Roodenburg, A. L.: Treatment of dermatitis due to chrysotherapy with ACTH, J.A.M.A., 146:1225, July 28, 1951.

4. Freyberg, R. H.: In Comroe's *Arthritis and Allied Conditions*, Hollander, J. L., editor: Gold Therapy for Rheumatoid Arthritis, Ch. 17:290, Lea and Febiger, Philadelphia, 1949.

THE CARE OF INFANTILE ECZEMA*

ALBERT V. STOESSER, M. D., Ph. D.†

MINNEAPOLIS, MINN.

There are many forms of therapy for eczema of infancy. In this outline an attempt has been made to present the more important and most practical features of all treatments. No effort has been made to go into any detail concerning the skin lesions or the handling of any complications.

SEBORRHEIC ECZEMA OR DERMATITIS

This is not a true allergic disease. It usually begins in the scalp and spreads to the face, involving the posterior aspect of the cheeks and the eyebrows. The lesions have yellow, greasy scales.

There is not a high incidence of allergy in the family; the cutaneous allergy tests are seldom positive, and the child does not develop other allergic manifestations later in life.

The skin may be cleansed with allercreme detergent oil, while liquid germicidal detergent (Parke Davis & Co.) or phisoderm with hexachlorophene (Winthrop) works well in the scalp. Boric acid ointment has been used on the face and scalp

*Presented at the Fourteenth Annual Meeting of the New Orleans Graduate Medical Assembly, New Orleans, March 7, 1951.

†From the Department of Pediatrics of the University of Minnesota Medical School.

but often it is not completely satisfactory, and the following may be tried:

℞ Resorcinal ..... 0.6 gm.
Velvachol ..... 60.0 gm.
(Texas Pharm. Co.)

℞ Salicylic-sulfur Ung. ..... 30.0 gm.
Velvachol ..... 30.0 gm.

℞ Vioform 3 per cent cream or ointment ..... 60.0 gm.

Diet is not important, although some investigators have suggested a reduction of fat. Skimmed evaporated milk is good. Dryco (Borden) or Alacta (Mead) have been used. Lately, Dalactum (Mead) has become quite popular.

ATOPIC ECZEMA OR ALLERGIC DERMATITIS

This allergic condition begins on the flushing areas of the cheeks and may spread to the forehead, ears, neck and extremities where it later is most marked in the cubital and popliteal folds. First, there is erythema, to be followed by papulovesicular eruption which itches. Scratching leads to oozing and crust formation.

The family history is usually positive for allergy. Cutaneous tests reveal positive reactions in approximately one-half of the cases. Egg white may be the only positive reactor. More often egg white and other foods, such as milk, rice, wheat, potato, tomato, or orange, give positive reactions. Occasionally, egg white does not react. In these cases, any positive reacting allergens are of clinical significance. One-third of the children with atopic eczema develop respiratory allergy (allergic rhinitis and/or asthma).

The skin may be cleaned with soap and water, *not an oil*. Lowila cake (Westwood Pharm. Co.) is preferred. However, phisoderm with hexachlorophene also may be employed.

When a cream or ointment is prescribed, orders should be given to apply them in the form of a thin layer, many times a day. As often as the infant rubs the preparations off, more is applied. This involves, in some instances, as many as ten applications in one day. The following have given good results:

℞ Bland cream ..... 60.0 gm.
(Dermovan of Texas Pharm. Co.)

℞ Calamine lotion (plain) ..... 120.0 cc
℞ Liquefied phenol ..... 0.1 to 0.5 cc.
Calotex (Texas Pharm. Co.) ..... 120.0 cc.

℞ Burrow's solution (Liq. alumin. acetate) prepared from domeboro tablets or powders and used as a continuous moist pack.

℞ Burrow's solution ..... 20 cc.
Velvachol ..... 20 to 30 gm.
Zinc paste (Lassar's) q. s. 60 gm.
} Modified Rosen's ointment

℞ Carbonis detergens cream of the Texas Pharm. Co. ..... 60 gms.

or

℞ Crude Coal tar ..... 0.5 to 1.0 gm.
Modified Rosen's Unq. ..... q. s. 60.0 gm.

℞ Crude coal tar ..... 0.5 to 1.0 gm.
Zinc oxide ..... 12.0 gm.
Amyli ..... 12.0 gm.
Velvachol ..... q. s. 60.0 gm.

Occasionally an infant will react to any tar preparation. This requires the substitution of naftalan

℞ Menthol ..... 0.6 gm.
Naftalan ..... 3.0 gm.
Burrow's solution ..... 15.0 gm.
Aquaphor, or Velvachol ..... 15.0 gm.
Zinc paste (Lassar's) ..... 60.0 gm.

℞ Ichthyol ..... 4.0 gm.
Naftalan ..... 20.0 gm.
Aquaphor, or Velvachol ..... 50.0 gm.
Zinc paste ..... q. s. 100.0 gm.

Contact with certain allergens is significant. Woolen clothing is not permitted and woolen blankets and other woolen bedding cannot be used. No feather stuffed pillows or mattresses are allowed. The mother should be instructed to keep the infant away from feather pillows on beds or couches about the home. Animals (cat and dog) should not come near the child. In this connection, some observers have stated that it is difficult to treat eczema on a farm. Dusty things must be avoided.

Trauma must be reduced. Cuffs may prevent the infant from bending the arms at the elbow and scratching the face. Sometimes further restraints are temporarily necessary. The arms and legs are then held outstretched by means of strips of cloth extending from the wrists and ankles to the bars on the crib. The skin at the wrist and ankle is well protected with cotton. If the child rubs the face on the bedclothes, a sheet of heavy weight plastic ma-

terial can be employed. A piece about 2 feet square is fastened to the bed under the infant's head by strips of adhesive tape.

Sedation is important. Elixir nembutal (Abbott) or amytal (Lilly) may be tried by giving one fourth teaspoonful every four to six hours, as necessary. Somnos (Sharpe & Dohme) has been most helpful. Occasionally, it becomes necessary to use chloral hydrate by rectum. The dose is 5 to 10 grains.

Antihistamines may act as sedatives. Syrup of histadyl, syrup of decapryn, and elixir benadryl have been worthwhile in some cases. To each dram of the latter may be added 1 8 of a grain of phenobarbital. This combination has been effective in the very irritable child.

The diet must not be forgotten:

1. *Breast fed infants*—The mother may omit all eggs and wheat (especially cereals) as much as possible, and reduce milk in her diet. Calciphos can be given to furnish a sufficient amount of calcium. All foods which cause gastrointestinal distress or urticaria must be avoided. The eczematous infant is fed every four hours, and 5 times in twenty-four hours, usually at only one breast, in order to prevent too rapid a gain in weight. Fat babies do not respond satisfactorily to present day methods of treatment.

2. *Artificially fed infants*—Again, the four hour schedule is followed. Evaporated milk formulas may be tried. If progress is not good, the feeding may be changed to evaporated goat's milk, nutramigen (Mead) or mull-soy (Borden). There is also Rowe's so-called meat milk.

Recently, much emphasis has been placed on the soy bean preparations which are usually well taken by infants and young children. Occasionally, there may be some gastrointestinal distress but this is overcome by adding 1 to 2 teaspoonfuls of kaopectate (Upjohn) or pectocel (Lilly) to each feeding.

3. *Elimination of certain food allergens* is essential. No infant should receive egg or egg products. Further removal of foods may be made on the basis of experience or may depend on the results of the cutaneous tests. Vitamins must be added in the form of adsorl and cecon (Abbott) or vi-penta (Hoffman-LaRoche). Iron is often necessary and it may be administered as zymatinic (Upjohn) or fer-in-sol (Mead) drops.

The treatment is broadened still more by the addition of lard or soybean oil. The oral administration of these fats increases the concentration of the serum unsaturated fatty acids and results in some cases in an improvement of the eczema. Offer 1 to 3 teaspoonfuls of the lard or 1 to 3 drams of the oil with each feeding.

ICTHYOTIC TYPE OF ECZEMA OR GENERALIZED SEBORRHEIC DERMATITIS

This disease is confused with erythroderma desquamativum (Leiner's disease). There is little involvement of the face, much of the extremities, and some of the trunk. Redness, thickening. induration, and scaliness are present. The scales are white and dry. Allergy may be present in the family. Positive cutaneous allergy tests are obtained. ACTH or cortisone may be used temporarily to clear the skin for skin testing. It must be remembered that these preparations cure nothing. Reactions to egg, milk, fish, and later in childhood, to nuts, are quite frequent. Some of the children develop respiratory allergy from the fish and nut sensitivity.

The skin should not be cleansed with mineral oil. Instead allercreme detergent oil or phisoderm with hexachlorophene may be employed. The sulfonated oil (Almay) without or with tar (5 per cent juniper tar) works well if there is much itching. The Almay tar bath, also, can be tried. It is better than the old colloidal baths. All ointments should contain very little petrolatum and the following topical therapy is suggested:

℞	Menthol	0.1 to 0.3 cc.
	Phenol	1.0 to 2.0 cc.
	Zinc oxide powder	10.0 gm.
	Lime water and oilve oil in equal parts to make	120.0 cc.
℞	Menthol	0.1 to 0.3 cc.
	Phenol	1.0 to 3.0 cc.
	Lubriderm (Texas Pharm. Co.)	120.0 cc.

℞	Phenol	1.2 cc.
	Zinc oxide	6.0 gm.
	Pulv. calamine	8.0 gm.
	Glycerine	10.0 cc.
	Milk of magnesia q. s. ad.	120.0 cc.
℞	Vioform cream (3 per cent)	60.0 gm.

℞ Olive oil with little chlorophyll, velvachol (Texas Pharm. Co.), geralatum (Almay), or aquaphor may keep the skin in good condition once the acute stage has subsided. Frequent application is necessary.

Itching is a prominent feature. It leads to much scratching with trauma, which greatly retards satisfactory progress. Creams and lotions containing antihistiminics are of little value. However, gelotion (Maroc Co.) has been helpful in some cases.

Diet regulation is important. Not only should egg be eliminated, milk substitutes employed, and other foods reduced, but fish and fish products avoided. Plenty of vitamins, especially A and D are indicated.

PYOGENIC OR INFECTIOUS ECZEMA

This resembles impetigo. Areas of eczema of a mild to moderate degree, located especially on the face, arms, and legs, have become infected. Very few pustules are present for the majority have been destroyed by scratching, and as a result there is much crusting.

Treatment is directed toward clearing-up of the infection. Years ago, the crusts were removed with a saturated solution of boric acid as a continuous wet pack, but now potassium permanganate in dilutions of 1-5000 to 1-10,000 is most popular. Following this procedure, aureomycin or terramycin ointment is indicated for a few days, after which bacitracin or tyroderm cream may be employed as long as necessary.

When the infection is gone, the eczema usually heals spontaneously. In this connection, it must be remembered that in the care of all eczemas, "Cleanliness is next to godliness."

ADDRESS OF THE PRESIDENT SURGICAL ASSOCIATION OF LOUISIANA 1950-1951

JAMES Q. GRAVES, M. D.

MONROE

It is with great pleasure and pride that I speak to you this evening as I relinquish the Presidency of this strong young Society into the capable hands of Dr. Jimmy Rives.

In these troubled times I have often liked to speculate as to the future of our profession, the profession of surgery. And like Patrick Henry, "I know of no way of judging the future but by the past." Let us look backward for a moment and see, in the perspective of time, where we, as surgeons, stand in the conflicting ideologies of today.

The discovery of anesthesia, in 1846, and asepsis, in 1867, made the surgeon a key figure in the great technologic development which followed during the last half of the nineteenth century. These two pivotal discoveries travelled to the four corners of the earth unimpeded by iron or silken curtains. New surgical techniques were developed by men of all nations, enriching all biological science. Pavlov was thus able to construct his gastric pouch, Sherrington to explore the nervous system, and Eck to devise his fistula.

The threshold of the twentieth century saw basically three categories of operative procedure: First, there were operations which had existed for centuries, such as vascular ligations, amputations of extremities, trephining and drainage of empyema, (ablations of superficial tumors, amputations of the breast, release of strangulated hernia, removal of vesical calculi.) Asepsis reduced the mortality rate of these procedures from 50 per cent to 1 to 2 per cent.

Second, were those surgical procedures which had been conducted from time to time but which were too dangerous before the advent of asepsis, such as ovariotomy, splenectomy, nephrectomy, operations for goiter, and plastic procedures, (intestinal

Address delivered at the Annual Meeting of the Surgical Association of Louisiana, New Orleans, November 11, 1951.

suture, opening of joints, repairs of non-strangulated hernia).

Third, were operations newly devised at the end of the nineteenth century, such as osteotomy, exploratory laparotomy, suture of the heart, and total hysterectomy, (laminectomy, transperitoneal nephrectomy, pyelotomy nephropexy).

Surgeons of the twentieth century have greatly extended the scope of operative surgery. So many changes have been made in old techniques and so many new techniques have been developed that only a few of the most outstanding ones can be mentioned here.

The most noteworthy advances in operative techniques have been in cardiovascular, pulmonary, and esophageal surgery. Other notable advances have been achieved in peripheral vascular surgery, plastic surgery, autonomic nervous system surgery, and in neurosurgery, especially of the brain.

The surgical procedures to correct congenital anomalies of the heart and major blood vessels probably should be placed at the top of the list. The results following operations on many disabling lesions are most promising. The correction of ductus arteriosus—a persistent patency of the vessel between the pulmonary artery and aorta as seen in blue babies—by ligation or division of this vessel has resulted in remarkable improvement in the condition of patients. Coarctation of the aorta—a narrowing or stricture of a portion of the thoracic aorta—is another anomaly which is now attacked by surgeons. In this operation the constricted area is resected and the normal vascular channel is restored or a bypass is established by anastomosing an adjoining blood vessel to the distal segment beyond the site of obstruction.

Surgical procedures have been developed also to repair defects in the septum of the heart. Patients with mitral stenosis can now be greatly improved by an operation in which a portion of the mitral valve is divided or resected, thereby creating a larger opening through which the blood flows from the auricle to the ventricle.

The most outstanding advance in general surgery during the past six years is the treatment of carcinoma of the thoracic esophagus and cardia of the stomach. Heretofore, the excision of a lesion in this portion of the esophagus resulted in a large defect which could not be bridged by any surgical procedure; the patient had to live as best he could with one end of his esophagus attached to the skin at the base of his neck.

At the present time the operation for carcinoma of the thoracic esophagus at any level is performed without hesitation. The continunity of the food passage is accomplished by bringing a portion of the stomach or the entire stomach through an incision in the diaphragm into the left pleural cavity and anastomosing the stomach to the divided end of the proximal esophagus. In low lying lesions this anastomosis can be accomplished below the transverse arch of the aorta; in high lying lesions the stomach is brought above and in front of the aorta and then the anastomosis is performed. In a few instances, in high lying lesions, the stomach has been brought completely up through the left chest and anastomosed to the esophagus just above the left clavicle in the base of neck.

The tremendous impetus to surgical technologic advance, begun in the last half of the nineteenth century has reached its culmination now. In twentieth century surgery, the emphasis is on sound, competent technology and, even more important, on the maintenance of the humanitarian qualities that are treasured so highly by responsible members of a great profession. The characteristic feature of the modern surgeon is his re-establishment as a biologist and as a physician in the broad sense of one skilled in the healing art. We are witnessing the beginning of the end of the sterile isolation of the surgeon at the operating table. We see the surgeon travelling in the company of others such as the internist, the physiologist, the chemist. We behold a common quest for elucidating unsolved problems in a free and democratic society.

Without restraint, modern surgery has advanced to where every cavity of the body is now accessible. Surgical skill, like sur-

gical science has made unparalleled progress second to no other science of this age.

But, what the next half century holds is now difficult to visualize. Due to the possibility of socialization, an interference, which is a factor for consideration, the perplexities of the future may be changed, not to the best interest of advanced surgery.

It would be rash, indeed, to try to foretell the progress of surgery for the next fifty years. In our zeal to fight the menace of socialization, the statism, we must not succumb to the danger of resisting all changes. We can lose this battle in the long run, if we fail to adopt a sound advance. Nothing in life stands still, we must march forward with progress or slide backward.

Progress is possible, only in a free society, rooted deep in democracy. If the responsibility for professional care should pass from the hands of the trained surgeon to the laymen, the status of the surgeon would inevitably be lowered to something resembling that of a hired technician. There would be no reward for skill, perseverance, and kindliness, nor for success in diagnosis and procedure. The motive, that now provides a positive incentive for higher quality of performance, would be turned in a negative direction.

We, surgeons of Louisiana, justly feel proud of the accomplishments achieved by science and join with the surgeons of the nation to prevent any political interference on the part of the evil of totalitarianism to destroy our freedom, for an uninterrupted interference, for the continuance of American progress, in surgery.

In the arena of medicine, as in industry, we have proved that the American way—the way of enterprise—worked best. If you allow that to be destroyed or weakened, through socialization, we will become as weak as Great Britain, France and Italy, which at one time were the most powerful nations on earth. Then, surely, we will be easy prey for Russia.

When the state enters the field of private activity, that field turns into a battleground of organized pressure groups, political and professional.

If we remain free and unrestrained in our scientific study to pursue the unsolved and intricate problems of surgery, there will be still greater benefits achieved for the betterment of the human race.

Gentlemen, in this changing world, the future of surgery lies in the hands of destiny. To safeguard its future is a challenge that we accept with full responsibility, without compromise.

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SOCIALIZED MEDICINE AS A CAMPAIGN ISSUE

State medicine is again an issue which is being discussed by the American public. It is fortunate that the sharp decision has been made possible. Democratic administration has been, and continues to advocate compulsory sickness insurance, which is State Medicine under another name. The Democratic platform says, in part, that the party advocates "federal aid for medical education to help overcome the growing shortages of doctors, nurses, and other trained health personnel." The party recognizes that "the costs of modern medical care have grown to be prohibitive for many millions of people. We commend President Truman for establishing the non-partisan commission on the health needs of the nation to seek an acceptable solution of this urgent problem."

From these and other statements, it is clear that the committee regards the field of medicine as legitimate ground for government direction and ultimately bureaucratic control. The statements of the Democratic party nominee give further credence to the belief that he advocates just such a program.

On the contrary, the Republican party platform gives the following statement:

> "We recognize that the health of our people as well as their proper medical care cannot be maintained if subject to federal bureaucratic dictation. There should be a just division of responsibility between government, the physician, the voluntary hospital, and voluntary health insurance. *We are opposed to federal compulsory health insurance with its crushing cost, wasteful inefficiency, bureaucratic dead weight, and debased standards of medical care.* We shall support those health activities by government which stimulate the development of adequate hospital services without federal interference in local administration. We favor support of scientific research. We pledge our continuous encouragement of improved methods of assuring health protection."

In accordance with the party platform, and obviously, his own belief in the matter, on September 14, General Dwight D. Eisenhower spoke emphatically against socialized medicine leading towards assembly line treatment of patients. He pointed out that the American people have the best medical care in the world today and indicated it would be a foolish experiment to attempt to nullify what our present system has already achieved. General Eisenhower further stated that he was opposed to a federally operated and controlled system of medical care, which is what the administra-

tion's compulsory health insurance scheme is in fact. He stated in so many words that we must "preserve the completely voluntary relationship between doctor and patient" with no intermediary, which is what the government would be if it paid the doctor. In his summary, he said:

> "Experience has shown that American medicine outstripped the world on a voluntary basis and on that basis—plus voluntary insurance plans, together with locally administered indigent medical care programs for those unable to participate—the needs of Americans will be most adequately met."

It is clear from this expression of thought that the Republican candidate has aligned himself clearly with the position on this issue which has long been that advocated by leaders of organized medicine.

The interests of the physicians as a whole over the nation, and particularly those in Louisiana, are at stake. The Louisiana primary law does not bind a party member in a national election. Every physician has the legal right to cast his vote for the national candidate of his choice. Physicians are urged to register, and urged equally strongly to vote so that there will not be an almost 20 per cent staying away from the polls as happened in 1948 in Louisiana.

The individual voter, in some instances, modestly seems to think that his vote does not count. Such self effacement seems to be mounting and riding at the heels of lethargy. Let us reflect that in recent years national legislation and even vicious trends in social and economic developments have been repeatedly put into being by compact, resourceful and aggressive minority groups. These movements would be powerless except for the apathy of the citizen. As a corollary to this, if the doctor is to maintain his right to practice he must no longer restrict himself to consideration of the affairs of the leukocytes, the antibiotics, and the disease pictures, but must assert himself as a citizen and as a member of organized medicine in every facet where his life touches that of the community. Reflection of this nature should leave no doubt as to what the physician should do as a citizen for the community and in the interests of better medicine.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

EXCERPT FROM ADDRESS BY PRESIDENT OF A. M. A.

Addressing the national convention of the American Legion in New York this week, Dr. Louis H. Bauer, president of the AMA, said it was heartening in this critical year of 1952 "to see that both of our major political parties—one of them directly and the other by implication—have recognized that great upsurge of public opinion on the issue of socialized medicine.

"The health plank in the Republican party platform," he continued, "includes the outright statement that 'We are opposed to Federal Compulsory Health Insurance with its crushing cost, wasteful inefficiency, bureaucratic dead weight, and debased standards of medical care.'

"The Democratic party platform—adopted by a convention representing the views of the party rank-and-file, in contrast to the views of a handful of planners in the executive department of the federal government—contains no endorsement of Compulsory Health Insurance, as has been the case with previous platforms, but simply urges a resolute attack against the financial problems involved in serious illness.

"In view of what has been happening during the past three or four years, both party actions confirm the validity of the medical profession's case against socialized medicine. The two party platforms also give reason for hope that never again will the people's health be used as a football in the political arena. Above all, however, they constitute a timely tribute to the forces of an aroused public opinion.

"The American Medical Association and the American Legion have much in common," he said. "Not only are they jointly interested in the health and welfare of the veteran, but they also have provided the main leadership in the drive against the socialism which is steadily creeping over this country."

Dr. Bauer said that American physicians will be forever grateful to the Legion for its early, vigorous and continued support against socialized medicine.

However, Governor Stevenson, in press conference and in public political addresses has stated that he has great confidence in Dr. Magnusson, his personal friend and Chairman of Mr. Truman's committee to study the subject of medical care in this country, and would await their report before stating his final opinion as to his stand on this subject. You can well appreciate, in advance, that this committee's report will to a certain degree, at least, be favorable to the wishes and feelings of Mr. Truman, who has consistently recommended and fought for socialized medicine. Beware of snares and traps.

General Eisenhower has expressed himself in unmistakable language that he is against socialized medicine. Why should we take chances or have any doubts when we go to the polls in November?

COMMISSION ON ACCREDITATION OF HOSPITALS

The new office of the Joint Commission on Accreditation of Hospitals at 660 Rush Street, Chicago, opened on September 1. Dr. Edwin L. Crosby, former superintendent of Johns Hopkins Hospital, Baltimore, is director of the Commission, which will assume responsibility for the hospital standardization program formerly carried out by the American College of Surgeons.

WAGE STABILIZATION

One of five physicians who practice medicine in a partnership and employ six persons to do office work inquired recently if the partnership was subject to restrictions imposed by Wage Stabilization.

Attorney T. V. McDavitt, director of the Industrial Relations department of the AMA, replied that under the circumstances, the partnership is not subject to Wage Stabilization and "the matter of wages and salaries paid to your employees is entirely within the discretion of the partners." He added that small business firms generally —those employing a total of eight or less persons in all branches—are exempt from wage and salary controls under the recent amendments to the Defense Production Act.

READING MATERIAL IN DOCTORS' OFFICES

Timothy Turner, columnist for the Los Angeles Times, wrote an article recently about the reading material usually found in doctors' offices. He studied the subject in detail and then came up with some ideas.

He said magazines for doctors' offices should be the ones that have short stories or articles. Continuing, he said:

"This leaves out such publications as the Saturday Evening Post and Collier's, for they have full-length articles and long short stories. One may have to wait for the doctor an hour or more, but hope springs eternal that he will not have to wait long, so he does not want to start a long story, for he may not be able to finish it.

"Here is my list: Newsweek, Reader's Digest, Life, Holiday, National Geographic, Harper's Bazaar, Vogue, Seventeen, Business Week, New Yorker, Esquire, and Today's Health (formerly Hygeia) an American Medical Association magazine."

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SYMPOSIUM ON CHILD HEALTH

A Symposium on Child Health will be held in Shreveport, November 15, 1952, from 9 A. M. to 12 Noon, at the Washington Youree Hotel. This will be held on the final day of the 1952 Annual Meeting of the Louisiana Health Council with the Caddo Health Council as host. The theme of the Health Council Meeting will be "Louisiana Plans for the Future of Public Health."

The Symposium will concern itself with "Advances in Medicine of General Interest in Regard to Children of School Age", with the folowing subjects being presented:

1. Nutrition.
2. Poliomyelitis.
3. Contagious Disease.
4. Accident Prevention.

A cordial invitation is extended to all doctors to attend the entire meeting, especially doctors from rural parishes and the smaller cities as the program is planned entirely for their benefit.

POST-GRADUATE COURSE ON THE CARE OF PREMATURES

A Post-Graduate Course on the Care of Prematures is being offered at the Charity Hospital of Louisiana, the Premature Infant Center, November 5-9, 1952. Registration is limited to ten and there will be no tuition fees. This course is open to all physicians and should be of particular interest to pediatricians and public health officers. Applications and request for further information may be addressed to :Dr. Elaine Allen, Premature Infant Center, Charity Hospital, New Orleans, La.

NEW LABORATORY OPENED

A new laboratory for testing devices submitted to the A.M.A. Council on Physical Medicine and Rehabilitation has been opened at A.M.A. headquarters. Most of the laboratory's work, done by Dr. Frederic T. Jung, is concentrated on testing the actual mechanics of new devices submitted by manufacturers to the Council. This supplements the clinical testing.

NEW OFFICE OPENS SEPT. 1

The new office of the Joint Commission on Accreditation of Hospitals, at 660 Rush Street, Chicago was opened on September 1. Dr. Edwin L. Crosby, former superintendent of Johns Hopkins Hospital, Baltimore, is director of the Commission, which will assume responsibility for the hospital standardization program formerly carried out by the American College of Surgeons.

SURGICAL ASSOCIATION OF LOUISIANA

The following program has been planned by the Surgical Association of Louisiana which will meet Sunday, November 16, 1952, at the St. Charles Hotel, New Orleans:

Dr. Cushman D. Haagensen, Institute of Cancer Recearch, Columbia University, College of Physicians and Surgeons, New York, New York—"Breast Cancer".

Dr. Willis J. Potts, The Children's Memorial Hospital, Chicago, Illinois—"Common Surgical Problems in Children".

Dr. W. R. Mathews, Shreveport Charity Hospital, Shreveport, La.—"Carcinoid Tumors of the Rectum".

Dr. Charles R. Walters, New Orleans, La.—"Problem in Diagnosis and Management of Gastrointestinal Bleeding".

Dr. Claude C. Craighead, New Orleans, La.—"Duodenal Fistula".

Business Meeting, Cocktails, Banquet.

INFORMATION REGARDING NEW ANTI-ARTHRITIC DRUG BUTAZOLIDIN®

(phenylbutazone)

At the meeting of the American Rheumatism Association held in Chcago June 6th and 7th, the first national reports were made on BUTAZOLI-

DIN (phenylbutazone) an important new agent for the relief of arthritis and allied disorders. As this meeting was open to the lay press, information about these reports was made available to reporters by the Committee on Public Relations of the Association, with the consequence that news items about the drug have appeared in certain periodicals. Care with regard to side effects was advised. The drug is contraindicated in severe renal, cardiac or hepatic disease.

RAPID STRIDES MADE IN EMERGENCY MEDICAL CALL PLANS

Rapid strides have been and are being made in the development of emergency medical call systems, the Council on Medical Service of the American Medical Association reported.

Today, approximately 80 per cent of the country's large medical societies have such plans, the council reported. In 1948, it pointed out, only about 60 medical societies reported having a formal plan for handling emergency and night calls. In 1949, the number of such plans rose to between 100 and 120.

At the end of 1951, 364 emergency medical call systems were in operation, according to the council. Of the 40 medical societies with 200 to 300 members, 29 had such plans, while 63 of the 71 medical societies with a membership in excess of 300 had such systems.

Despite such impressive progress, the council urged all counties where such plans were not in operation to begin developing one immediately.

"Development of an efficient emergency call plan is an important step any medical society can take toward assuring the public that medical care will be available when needed," it stated.

"While every county or city has real emergency cases, not every county medical society needs a formal or organized program for handling emergencies."

PARENTS MUST CONDITION CHILD EMOTIONALLY FOR OPERATION

Preparation of a child for surgery must begin in the home by the provision of an environment of love, trust and security if the child is to survive the operation without suffering emotional injury.

This conclusion was expressed in an article in the Journal of the American Medical Association written by Katherine Jackson, M. D.; Ruth Winkley, A. B.; Otto A. Faust, M. D., and Ethel G. Cermak, M. D., all of Albany, N. Y. Drs. Jackson, Faust and Cermak are associated with the departments of pediatrics and anesthesiology, Albany Medical College.

"Our experience has shown that the child best able to meet the hospital-anesthesia-surgery situation is the child who is able to trust the physicians and nurses to treat him fairly," the authors stated. "He assumes that painful procedures are necessary, are for his own good, and are not punitive."

ONE OUT OF THREE BOXERS MAY HAVE BRAIN DISORDER

One out of every three boxers medically examined under Colorado state athletic commission regulations was found to have abnormal brain wave recordings, possibly indicative of brain damage, according to a report in the August 23 Journal of the American Medical Association.

The compulsory brain examinations, by means of an electrical brain wave recording apparatus (an electroencephalograph), had been ordered by the commission as another move to prevent serious injury or death from the sport.

During a year's time, 24 boxers underwent such examinations in Colorado. The electroencephalogram records of four boxers showed severe disturbances and five showed moderate disorders, a total affected rate of 37.5 per cent, according to Drs. Elwald W. Busse and Albert J. Silverman, of Denver. The doctors are associated with the Colorado Psychopathic Hospital.

THIS IS THE RECORD

When it comes to exercising their voting privileges, the citizens of many other nations are way ahead of those of the United States. The percentage who went to the polls is revealed by these startling and astonishing facts:

In Belgium, March, 1950, 90 per cent.

In Italy, April, 1948, 89 per cent.

In England, October, 1951, 83 per cent.

In Canada, June, 1949, 75 per cent.

In Israel, July, 1951, 72 per cent.

In Sweden, September, 1951, 80 per cent.

In France, October, 1945, 75 per cent.

In Japan, June, 1950, 71 per cent.

In the United States, in the last presidential election, only 51 per cent of the eligible voters went to the polls; Forty-nine per cent of those eligible did not take the trouble to vote!

This is a year of decision. Every man or woman who is eligible to vote is urged to exercise his high privilege as a citizen—and vote.

VOTE AS YOU PLEASE . . .BUT
PLEASE VOTE

From The Missouri Pacific Lines "News Reel"

PRIMAQUINE, NEW DRUG, PREVENTS MALARIA RELAPSE

Announcement is made in the August 23 issue of the Journal of the American Medical Association of the development of a new antimalarial, primaquine, which destroys the parasite causing relapsing malaria and is cited by U. S. Army medical authorities as the most effective preparation known for the prevention of relapses.

Reports on the new compound and on its therapeutic efficacy are contained in a series of four papers in the AMA Journal. The reports cover studies conducted by teams of malaria experts working under the sponsorship of the Office of the Surgeon General, U. S. Army, and the U. S. Public Health Service.

IN MEMORIAM

Whereas, Dr. John G. Snelling was a member in good standing in the Ouachita Parish Medical Society.

Whereas, he was a valuable asset to the medical profession and whereas he was a valuable citizen in our community.

Be it therefore resolved that the Ouachita Parish Medical Society has lost a valuable member.

Further be it resolved that the society wishes to express our sincere sympathy and deep regrets to the family of Dr. John G. Snelling as a result of his passing.

William L. Bendel, M. D.
Chairman of Committee
Ben Cobb, M. D.
Roy A. Kelly, M. D.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

AUXILIARY TO SHREVEPORT MEDICAL SOCIETY

The annual meeting of the Woman's Auxiliary to the Shreveport Medical Society was held May 14, 1952, in the home of Mrs. R. T. Lucas.

The main item of business was the election and installation of the following officers:

President, Mrs. Paul D. Abramson; President-elect, Mrs. C. E. Boyd; First vice-president, Mrs. G. A. Creel; Second vice-president, Mrs. J. E. Knighton, Jr.; Recording Secretary, Mrs. J. E. Carlisle; Corresponding Secretary, Mrs. C. S. Holt; Treasurer, Mrs. E. Clay Edwards.

Mrs. Worley, retiring president, presented the gavel to Mrs. Abramson, recalling that the gavel had been presented to the Auxiliary, in 1943, by a former president, Mrs. B. C. Garrett. The names of each succeeding president were read. Mrs. Abramson graciously received the gavel,—and reported on the chairmen of committees for the coming year.

A most pleasant social hour was enjoyed, and the group adjourned to meet again in October.

LAFAYETTE PARISH MEDICAL AUXILIARY

Mrs. Thomas A. Kimbrough was installed as president of the Lafayette Parish Medical Auxiliary at the May luncheon meeting of the group at Jacob's Four Corners restaurant.

Other officers for the coming year are: president-elect, Mrs. J. R. Ferguson; secretary, Mrs. D. N. Chesson; treasurer, Mrs. Paul Kurzweg, Jr. . Mrs. L. B. Long, retiring president, thanked each officer and all the members for their work and fine cooperation during the past year.

Mrs. Kimbrough named all the committees for the coming year. She also made a report on the state medical convention recently held in Shreveport. It was announced that Mrs. J. Boring Montgomery of the Lafayette auxiliary was elected fourth vice-president of the State Medical auxiliary.

It was announced that representatives from the Lafayette Parish Medical auxiliary are now visiting all of the high schools in the parish in the interest of nurse recruitment.

The president announced that this was the last meeting of the auxiliary until September.

Other members present in addition to the new officers were Mrs. L. B. Long, Mrs. Michael Boustany, Mrs. M. E. Saucier, Mrs. Thomas Latiolas, Mrs. L. A. Prejean, Mrs. Ralph Bourgeois, Mrs. Edward Harrell, Mrs. Edward Wynne, Mrs. C. K. Oliver, Mrs. J. O. Duhon, Mrs. James Gilly, Mrs. Henry Voorhies, Mrs. W. L. Zinc, Mrs. John Miles, Mrs. F. H. Davis, Mrs. L. O. Clark, Mrs. Sidney Hernandez, Mrs. Mims Mitchell, Mrs. J. B. Montgomery, and Mrs. J. J. Burdin.

Honorable metnion was given three Lafayette parish high school students in the state judging of an essay contest sponsored by the American Medical Association. Students given honorable mention were Miss Joy Phillips, Lafayette High School, Miss Martha Briggs, Mt. Carmel High School, and Miss Shirley Billeaud, St. Cecilia High School of Broussard. The title of the essays was "Why the Private Practice of Medicine Furnishes this County with the Finest Medical Care." The essay contest sponsored by the American Medical Association was handled by the Lafayette parish Medical Auxiliary.

OFFICERS AND CHAIRMEN — WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY 1952-53

OFFICERS

President—Mrs. T. E. Strain, 743 Thora, Shreveport.

President-Elect—Mrs. Edwin Socola, 1216 Broadway, New Orleans.

1st Vice-President—Mrs. A. Scott Hamilton, 1300 Island Drive, Monroe.

2nd Vice-President—Mrs. J. E. Sorells, 115 College St., Lake Charles.

3rd Vice-President—Mrs. D. B. Barber, Box 446, Pineville.

4th Vice-President—Mrs. J. Boring Montgomery, 402 Jefferson Blvd., Lafayette.

Recording Secretary—Mrs. M. E. Kopfler, 750 Moore St., Baton Rouge.

Treasurer—Mrs. Dorf Bean, 615 Oneonta, Shreveport.

Corresponding Secretary—Mrs. Lucius L. Davidge, 4141 Richmond, Shreveport.

Parliamentarian—Mrs. A. A. Herold, 731 Oneonta, Shreveport.

CHAIRMEN

Archives—Mrs. C. Grenes Cole, 4938 Ct. Charles, New Orleans.

Bulletin—Mrs. Blaise Salatich, 24 Warbler, New Orleans.

Cancer Control—Mrs. Louis Leggio, 8300 Sycamore, New Orleans.

Commemoration Fund—Mrs. Cyril T. Yancy, 1704 N. Fourth, Monroe.

Doctor's Day—Mrs. P. R. Gilmer, 851 Olive St., Shreveport.

Editor of Publications—Mrs. O. B. Owens, 1026 Bolton, Alexandria.

Finance—Mrs. H. Theodore Simon, 1300 Third St., New Orleans.

Historian—Mrs. Clarence Webb, 3904 Creswell, Shreveport.

Today's Health—Mrs. Robert L. Simmons, 35 Fontainbleau Dr., New Orleans.

Legislation—Mrs. J. E. Carlisle, 358 Albany Ave., Shreveport.

Press and Publicity—Mrs. Leon Gray, 1901 Centenary, Shreveport.

Printing—Mrs. W. A. McBride, 213 Pennsylvania, Shreveport.

Essay Contest—Mrs. F. C. Shute, Opelousas.

Red Cross—Mrs. Jacob O. Hoth, 1378 Richland, Baton Rouge.

Romance and Research of Medicine—Mrs. James W. Warren, 470 Audubon Blvd., New Orleans.

Program—Mrs. Wiley A. Dial, 1137 Park Blvd., Baton Rouge.

Public Relations—Mrs. DeWitt T. Milam, 1704 Island Dr., Monroe.

Revision of By-Laws—Mrs. W. B. Worley, 558 Longleaf Dr., Shreveport.

Year Book—Mrs. John L. Beven, 783 Lakeland Dr., Baton Rouge

Nurse Recruitment—Mrs. Ralph Talbot, 200 Arkansas, Monroe.

La. Health Council—Mrs. M. C. Wigginton, Hammond.

Civil Defense—Mrs. W. A. K. Seale, 221 W. Elizabeth St., Sulphur.

COUNCILORS

First District—Mrs. Nathan Polmer, 2207 Carondelet, New Orleans.

Second District—Mrs. Albert F. Habeeb, 178 W. Oakridge, New Orleans.

Third District—Mrs. Isadore W. Gajan, New Iberia.

Fourth District—Mrs. W. J. Hill, 725 Erie, Shreveport.

Fifth District—Mrs. A. G. McHenry, 1810 Riverside Dr., Monroe.

Sixth District—Mrs. Rhodes J. Spedale, Plaquemine.

Seventh District—Mrs. John G. McLure, Welsh.

Eighth District—Mrs. D. B. Barber, Box 446, Pineville.

PARISH PRESIDENTS 1952-53

First District—Orleans

Mrs. Louis Leggio, 8300 Sycamore St., New Orleans.

President-Elect—Mrs. Edwin R. Guidry, 720 Broadway, New Orleans, to take office in January.

Second District

Mrs. J. J. Massony, Westwego, La.

Third District

Lafayette

Mrs. Thomas Kimbrough, 306 St. Thomas St., Lafayette.

Lafourche

Mrs. Thomas B. Ayo, Raceland, La.

Terrebonne

Mrs. Buford J. Autin, 235 Sunset Avenue, Houma, La.

Fourth District—Caddo

Mrs. Paul D. Abramson, 704 McCormic, Shreveport, La.

Fifth District—Ouachita

Mrs. J. W. Schonlau, 1507 Spencer Street, Monroe, La.

Sixth District—East Baton Rouge

Mrs. Henry W. Jolly, Jr., 1420 Stanford Avenue, Baton Rouge

Iberville

Mrs. Rhodes Spedale, Plaquemine, La.

St. Tammany

Mrs. H. E. Cannon, Covington, La.

Tangipahoa

Mrs. I. I. Rosen, Amite, La.

Washington

Mrs. Lamar Lancaster, 621 Huron, Bagalusa, La.

Seventh District—Beauregard

Mrs. Luke Marcell, 602 S. Royal Street, DeRidder, La.

Calcasieu

Mrs. Walter Moss, 2002 Drew Park Drive, Lake Charles, La.

Jefferson Davis

Mrs. L. E. Shirley, Jennings, La.

Iberia

Mrs. L. M. Villien, Jeanerette, La.

St. Landry

Mrs. S. J. Rozas, Opelousas.

Eighth District—Rapides

Mrs. N. N. Brian, Jr., 2000 Donahue Ferry Rd., Pineville, La.

SOUTHERN MEDICAL AUXILIARY INVITES WIVES TO MIAMI

The Southern Medical Association meets in Miami, Fla., November 10-13, 1952, and all indications are that it will be a meeting to be long remembered. The hospitable Miamians are planning a delightful social program for the ladies.

(A tentative) Auxiliary program is as follows:

Sunday, Nov. 9—Special Executive Committee meetings.

Monday, Nov. 10—Luncheon for Past Presidents. Luncheon for Councilors.

Tuesday, Nov. 11—Executive Board Breakfast; General Session; Doctors Day Luncheon; Other social activities, including a fish fry on the beach.

Wednesday, Nov. 12—General Sessions; Luncheon honoring the president, Mrs. V. Eugene Holcombe, the President-Elect, Mrs. Richard F. Stover and visiting State Presidents and Charter Members.

Thursday, Nov. 13—Executive Board Banquet.

The Auxiliary to the American Medical Association will furnish two of the speakers. Mrs. Ralph B. Eusden, President of the Auxiliary to the A. M. A., will discuss the aims and general program of the Auxiliary and Mrs. John McCuskey, a vice Chairman, will speak on nurse recruitment.

Wives attending the Southern Medical Association meeting with their husbands are cordially invited to attend all activities of the Auxiliary.

All reservations should be made early.

WOMAN'S AUXILIARY TO THE SOUTHERN MEDICAL ASSOCIATION

A notice from the Auxiliary to the Southern Medical Association states that the auxiliary is proud to have on its Executive Board this year the following outstanding members from Louisiana:

Mrs. Wiley R. Buffington, New Orleans; Mrs. Arthur A. Herold, Shreveport; Mrs. R. M. Simonton, State Councilor, Monroe; Mrs. Dewitt Milam, State Vice Councilor, Monroe.

BOOK REVIEWS

Surgical Practice of the Lahey Clinic: By the Staff of the Lahey Clinic. Philadelphia. W. B. Saunders Company, 1951. pp. 1014. Price $15.00.

In this second edition of the Surgical Practice of the Lahey Clinic are explained and demonstrated in 1,014 pages and 784 illustrations the operative techniques currently in use at the Lahey Clinic. Fully as valuable as the description of operative procedures is the thorough discussion of current diagnostic methods, preoperative and postoperative care, anesthetic procedures, concurrent medical treatment, and evaluation of end-results.

The wide variety of topics considered are: Thyroid and Neck (172 pages); Thoracic Surgery (93 pages); Abdominal Surgery (233 pages); Breast (10 pages); Gynecology (29 pages); Bones and Joints (33 pages); Neurosurgery (90 pages); Anesthesia (58 pages); and Miscellaneous Subjects (48 pages).

WALTER F. BECKER, M. D.

Brain Tumors of Childhood: By Henry M. Cuneo, M. D., and Carl W. Rand, M. D. p. 224. Charles C. Thomas, Publisher, 1952. Price $5.75.

This clearly printed monograph of the American Lecture Series presents a compact, well illustrated summary of current opinion on diagnosis, life history and treatment of most of the intracranial tumors occurring under 13 years of age. It is directed to both neurosurgeons and pediatricians and follows the pattern set by Cushing in presenting 2000 tumors in 1932. In a sense, it is a short sequel to the more encyclopedic presentation in 1939 by P. Bailey, D. N. Buchanan and P. C. Bucy. Discussion of the pathology of gliomas is brief, but other lesions are discussed more fully.

Eighty intracranial tumors encountered at the Children's Hospital in Los Angeles in the past decade were analysed together with three eosinophilic granulomas of the calvarium seen with them. The usual incidence of 74 per cent gliomas was reported among the intracranial lesions. Sixty-eight were operated upon with 19 deaths related to operation either immediately or in the next few weeks. No deaths occurred from ventriculography, but emphasis is laid upon 6 deaths arising from the consequences of failing to employ this study and the attendant erroneous exploration of the posterior fossa for a third ventricle tumor. Thirteen died with unfavorable lesions either from the point of view of position or histopathology. No deaths occurred among 3 infectious granulomas, and 1 optic nerve glioma. Among 17 cerebellar astrocytomas there was no immediate operative mortality al-

though one of these died two months postoperatively of streptococcus meningitis. An additional case of ependymoma also died of postoperative infection.

Five year cures were fewer than expected from previous experience particularly with cerebellar astrocytomas. The five year survivals reported included 1 infectious granuloma, 1 of 13 surviving with medulloblastoma, 2 of 9 surviving operation with craniopharyngioma and 3 patients with cerebellar astrocytoma of 16 surviving operation. Unfortunately, 26 of 49 patients surviving operation were lost from observation so that the results were incomplete. Of these, 10 were cerebellar astrocytomas. In view of 15 five year cures among 39 similar cases reported by Cushing in 1930 (28 patients were still living at the time of the report) it seems likely that many of the 10 cases lost track of survived five years at least. Recurrence and reoperation were not reported. This might be attributed to complete removal of mural nodules which is more common since Cushing emphasized its importance.

W. Randolph Page, M. D.

Clinical and Roentgenologic Evaluation of the Pelvis in Obstetrics; by Howard C. Moloy, M. D. Philadelphia, W. B. Saunders Co., 1951. pp. 119. illus. Price $2.50.

This is the first of a series of monographs designed to present in a somewhat condensed form some of the practical results of research in special fields of medicine. The author is an expert in roentgenologic pelvic mensuration and its obstetrical interpretation. The subject is presented in 116 pages of easily readable material with numerous explanatory diagrams, photographs, and pelvic x-rays.

A fine effort has been made in the description of pelvic morphology, including a complete but concise analysis of all anatomic types and their variations. The relationship to constitutional habitus is covered and a complete pelvic classification is presented.

A clinical pelvic examination is included, in addition to basic summaries of fetal relationship, mechanism of labor, forceps problems and breech delivery.

The various roentgenologic techniques and their interpretations are then described and the methods given for quantitative estimation of cephalopelvic disproportion.

This monograph seems primarily designed for the students of obstetrics but is certainly a valuable aid to the obstetric practitioner. Of course, the majority of obstetric opinion still holds that there is no substitute for clinical judgment and experienced obstetric intuition, and that it can never be replaced by x-ray analysis. However, expert roentgenologic aid has certainly been proven a worthwhile adjunct in combating the problem of difficult labor.

George T. Schneider, M. D.

Chronology of Ophthalmic Development; by Arthur H. Keeney, M. D. Springfield, Ill., Charles C. Thomas, 1951, pp. 32, charts 3, figs. none. Price $2.00.

This publication in the *American Lecture Series* (Ophthalmology, ed. Donald J. Lyle) is aptly described by its subtitle: "An outline summary of the anatomical and functional development of the visual mechanism before and after birth." The main part of the work consists of three large folding charts which list the time-tables of development of the principal ocular and accessory structures: orbit and general considerations; extra-ocular vascular system; nerve supply and extra-ocular muscles; lens and capsule; optic nerve; hyaloid system and retinal circulation; vitreous and suspensory ligament; retina; macula; choroid; lids and lacrimal apparatus; cornea; iris and ciliary body; sclera and Schlemm's canal. Two charts are devoted to the prenatal stages and the third carries on the development from the neonatal period through twenty to twenty-five years. The format of the charts is convenient for reference, with vertical columns for the structures named above and horizontal divisions for the successive ages. The descriptive entries are concise. As the preface states, "An understanding both of the general processes of embryology and of the organogenesis of the eye is presupposed." With such background, however, or while gaining it, this monograph provides a time-table summary that will be useful. It should be of particular service in considering congenital developmental faults.

Harold Cummins, Ph.D.

Amenorrhea; by Lawrence M. Randall, M. D. and Thomas W. McElin, M. D. Springfield, Ill., Charles C. Thomas, 1951. pp. 81. Price $2.25.

This monograph is one of a series of lectures covering the entire field of endocrinology and is edited by Willard O. Thompson. The subject of amenorrhea is covered more or less completely by the authors in that they have reviewed the entire literature on the subject during the years 1945 to 1949, inclusive.

The presentation is divided into sections, including a classification and definition of terms, a discussion of the phenomena of war amenorrhea with its hypothalamic and nutritional influences, and a broad review of treatment. In the latter is included a discussion of the use of thyroid, estrogen, progesterone, gonadotropins, x-ray, and various miscellaneous factors.

The authors express very little of their own opinion regarding their preferences in treatment and the reader is allowed to draw his own conclusions. For this reason, the monograph will be of small

practical value to the practitioner; but to the individual who is interested specifically in the various world viewpoints of the etiological background and the modern therapeusis of amenorrhea, it is of inestimable value.

GEORGE T. SCHNEIDER, M. D.

Doctors Differ; by Harley Williams. Springfield, Ill., Charles C. Thomas, 1952. 239 pp. illus. Price $7.50.

Five physicians, John Elliotson, Hugh Owen Thomas, James Mackenzie, William Macewen and R. W. Philip are presented in this study in contrasts. The author shows in an authoritative, but nontechnical way why doctors differ from one another and how new approaches, ideas, and methods of treatment came out of this difference and clash in personality. This is a book of individual biographies and shows that differing among doctors arises practically from differences in human make-up.

Chapters are devoted to John Elliotson, a London professor who quarrelled with other doctors over mesmerism and became the unacknowledged pioneer of psychological healing in this country; Hugh Owen Thomas, who founded modern orthopedics; Sir James Mackenzie, the heart specialist in contrast to Sir William Osler; Sir William Macewen in contrast to Sir Victor Horsley a brilliant pair of opposites who brought brain surgery to success; and lastly, Robert William Philip and Edward Livingstone Trudeau in the conquest of tuberculosis.

The book comes to a close with the epilogue: Rembrandt's picture, a sombre painting called "The Anatomy Lesson" by Dr. Nicholas Tulp. Our five doctors are watching this demonstration each thinking his own thoughts and representing five different philosophies each one true.

This book is well written and well worth reading.

RUTH E. HARLAMERT.

Doctors in Blue; a Medical History of the Union Army in the Civil War; by George Worthington Adams. New York, Henry Schuman, 1952. pp. 253. illus. Price $4.00.

It is interesting that this study on the Medical Service of the United States Army in the War between the States, should have been written by a Professor of History, rather than a physician. Dr. Adams has taught at Massachusetts Institute of Technology and at Harvard University. He is presently at Colorado College.

Dealing with the period 1860-65, the book describes the dire emergency presented to a small and poorly organized medical service, by the outbreak of this War. Medical knowledge and techniques are of course those of the period and are known to physicians; new and timely, however, is the story of the development and organizational plan of the Medical Service, from which has come the unexcelled health care given our soldiers today. The book is a valuable addition to American medical history.

MARY LOUISE MARSHALL.

Neurosurgery: An Historical Sketch; by Gilbert Horrax, M. D., Sc. D. Illus. Charles C. Thomas, Springfield, Ill. pp. 135. Price $3.75.

Few neurosurgeons are as well qualified as the author to present such an intimate view of the specialty. As one who has devoted a profitable lifetime to the field after long association with Harvey Cushing, he uses his broad background of personal experience to advantage. An authoritative view of many of the trends in the field is provided from the earliest knowledge to the present. It is lucidly written and of considerable general interest. Neurological will find much interesting and essential material although progress of the past decade is surveyed briefly.

W. R. PAGE, M. D.

PUBLICATIONS RECEIVED

Doubleday & Company, Inc., N. Y.: Living with Cancer, by Edna Kaehele.

Paul B. Hoeber, Inc., N. Y.: Cardiac Therapy, by Harold J. Stewart, M. D.; Physical Foundations of Radiology, by Otto Glasser, Edith H. Quinby, Lauriston S. Taylor, and J. L. Weatherwax (2nd Edit.).

Lange Medical Publications, Los Altos, Calif.: Physician's Handbook, by Marcus A. Krupp, M. D., Norman J. Sweet, M. D., Ernst Jawetz, M. D., and Charles D. Armstrong, M. D., (7th Edit.).

J. B. Lippincott Company, Phila.: Essentials of Dermatology, by Norman Tobias, M. D., (2nd Edit.); Medical Licensure Examinations, edited by Walter L. Bierring, M. D., (7th Edit.); Viral and Rickettsial Infections of Man, edited by Thomas M. Rivers, M. D., (2nd Edit.).

Medical Research Press, N. Y.: Sex After Forty, by S. A. Lewin, M. D., and John Gilmore, Ph. D., with Introduction by The Rev. Dr. Russell L. Dicks.

W. B. Saunders Co., Phila.: Gynecologic and Obstetric Pathology, with Clinical & Endocrine Relations, by Emil Novak, M. D., (3rd Edit.).

Charles C. Thomas, Publisher, Springfield, Ill.: Sterility, its Cause and its Treatment, by J. Jay Rommer, M. D.; Intracranial Aneurysms, by Wallace B. Hamby, M. D.

Von Hoffmann Press, Inc., St. Louis: Research in Endocrinology, by August A. Werner, M. D., and Associates, edited by Al. R. Schmidt.